CHAPTER 1

Eyelids

Applied anatomy

1. **The grey line** is an important structure because it divides the eyelids into an anterior lamella, composed of skin and orbicularis, and a posterior lamella consisting of tarsal plate and conjunctiva (Fig. 1.1).
2. **Glands** in the lid margin may be the source of cyst formation and occasionally tumours.
   a. *Meibomian glands* are modified sebaceous glands located in the tarsal plate and secrete the outer lipid layer of the precorneal tear film.
   b. *Glands of Zeis* are modified sebaceous glands that are associated with lash follicles.
   c. *Glands of Moll* are modified sweat glands the ducts of which open either into a lash follicle or directly onto the anterior lid margin between the lashes.
3. **The lashes** are slightly more numerous in the upper (approximately 100) than in the lower lid. The lash roots lie against the anterior surface of the tarsus in the space between the pretarsal orbicularis oculi muscle and the muscle of Riolan. The lashes pass between the orbicularis oculi and the muscle of Riolan and exit the skin at the anterior lid margin. All lashes curve away from the globe and are relatively parallel. Because they lack erectores pilorum muscles, their position and direction are determined by the surrounding orbicularis oculi, muscle of Riolan and tarsal plate. Thus, if the tarsus or orbicularis is abnormal, lash position and direction may be affected.
4. **Upper lid elevators** (Fig. 1.2)
   a. *The levator aponeurosis* fuses with the orbital septum about 4 mm above the superior border of the tarsus. Its posterior fibres insert into the lower third of the anterior surface of the tarsus. The medial and lateral horns are expansions which act as check ligaments. Surgically, the aponeurosis can be approached through the skin or the conjunctiva.

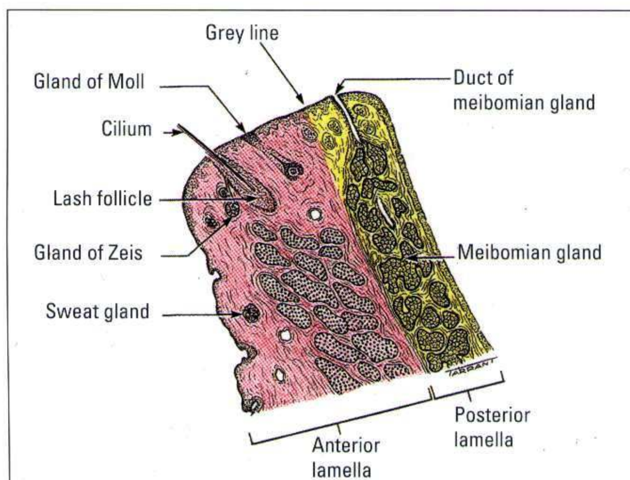


Fig. 1.1
Cross-section of the lower lid

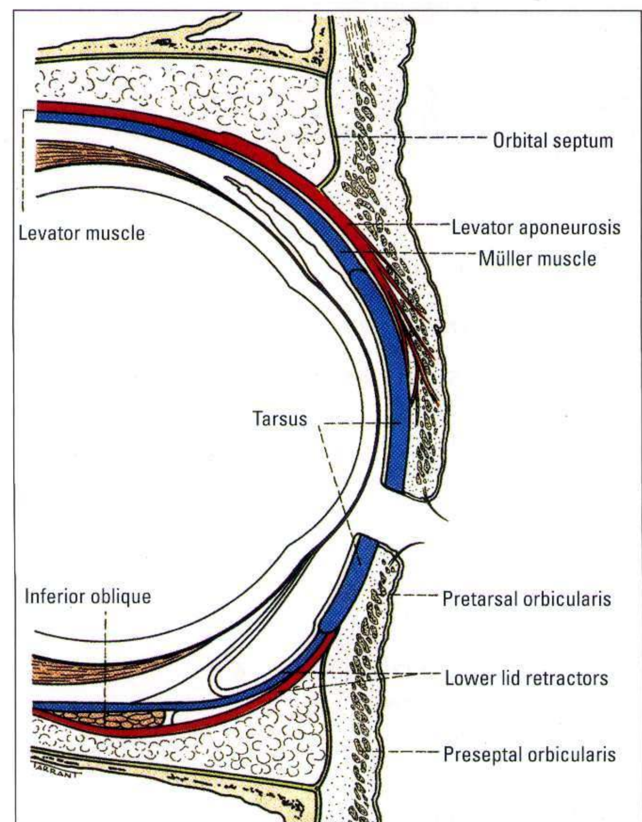


Fig. 1.2
Anatomy of the levator complex and lower lid retractors

   b. *Müller muscle* is inserted into the upper border of the tarsus and can be approached transconjunctivally.
5. **Lower lid retractors**
   a. *The inferior tarsal aponeurosis* consists of the capsulopalpebral expansion of the inferior rectus muscle and is analogous to the levator aponeurosis.

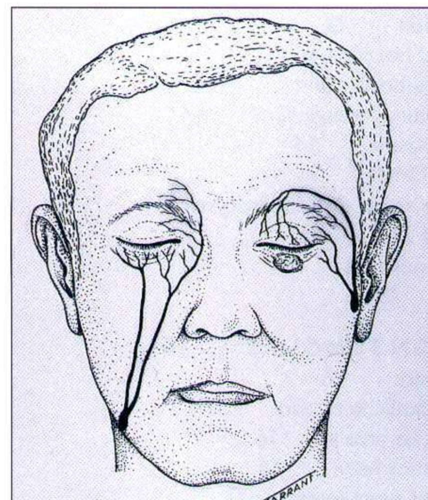


Fig. 1.3
Lymphatic drainage of the lids

b. *The inferior tarsal muscle* is analogous to Müller muscle.
6. **Lymphatic drainage** (Fig. 1.3). The upper lid and lateral canthus drain into the preauricular nodes, whereas the lower lid and medial canthus drain into the submandibular nodes.

Disorders of lashes

Trichiasis

Trichiasis is a very common acquired condition which may occur in isolation or as a result of scarring of the lid margin secondary to chronic blepharitis, herpes zoster ophthalmicus and trachoma. Trichiasis should not be mistaken for pseudotrichiasis secondary to entropion because in some cases the inturning of the eyelid may be intermittent and the condition may be mistaken for true trichiasis and inappropriately treated.

Signs

Posterior misdirection of lashes arising from normal sites of origin (Fig. 1.4). Trauma to the corneal epithelium may cause punctate epithelial erosions and ocular irritation made worse on blinking. Corneal ulceration and pannus formation may occur in severe long-standing cases.

Treatment

1. **Epilation** with forceps is simple and effective but recurrences within 4–6 weeks are inevitable.
2. **Electrolysis** is useful for a few isolated lashes but is tedious and frequently multiple treatments are required to obtain a satisfactory result. An electrocautery needle is inserted down the shaft of the lash root and current applied until coagulated tissue bubbles to the surface (Fig. 1.5). The lash is then removed. Retreatment for recurrences is required in about 40% of cases and can cause scarring.
3. **Cryotherapy** is very effective in eliminating many lashes (Fig. 1.6). With a special cryoprobe a double freeze–thaw cycle at −20°C is applied. Potential complications include skin necrosis, depigmentation in dark-skinned individuals, damage to meibomian glands, which may adversely affect the precorneal tear film, and shallow notching of the lid margin.
4. **Argon laser ablation** is useful for a few scattered lashes and is performed as follows:
   a. The initial settings are 50 μm, 0.2 seconds and 1000 mW.
   b. The laser is fired at the root of the lash and a small crater formed.



Fig. 1.4
Trichiasis

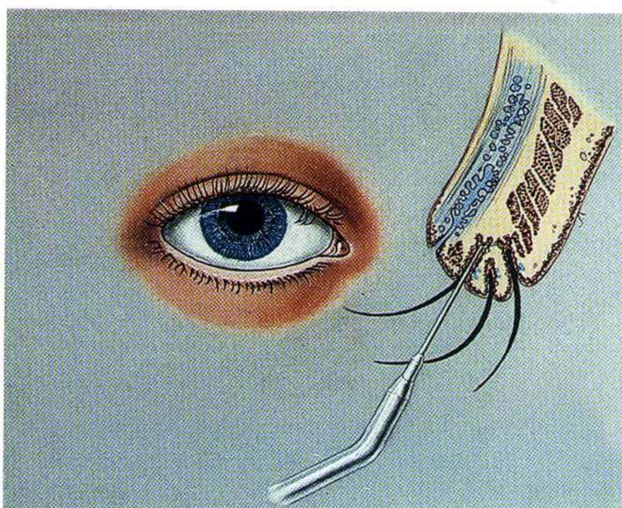


Fig. 1.5
Treatment of trichiasis by electrolysis (Courtesy of Wilmer Institute)

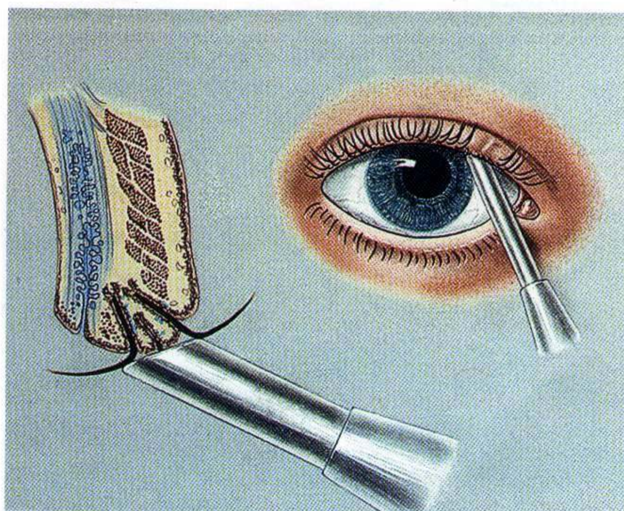


Fig. 1.6
Treatment of trichiasis by cryotherapy (Courtesy of Wilmer Institute)

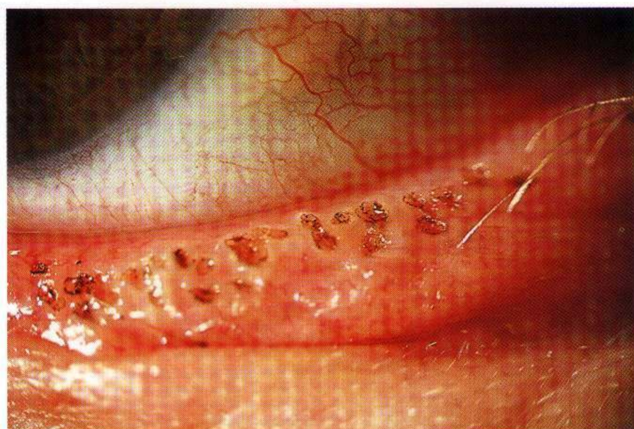


Fig. 1.7
Appearance following laser ablation of trichiasis

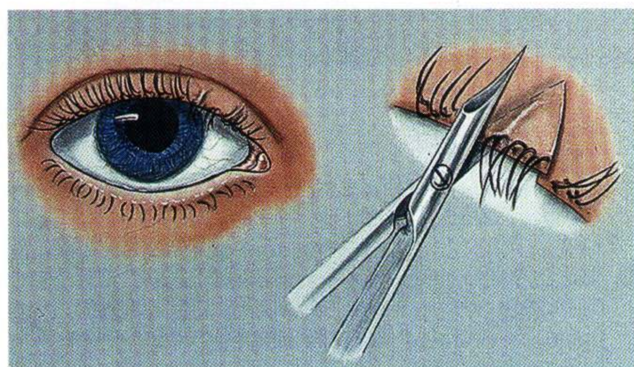


Fig. 1.8
Treatment of trichiasis by full-thickness wedge resection (Courtesy of Wilmer Institute)

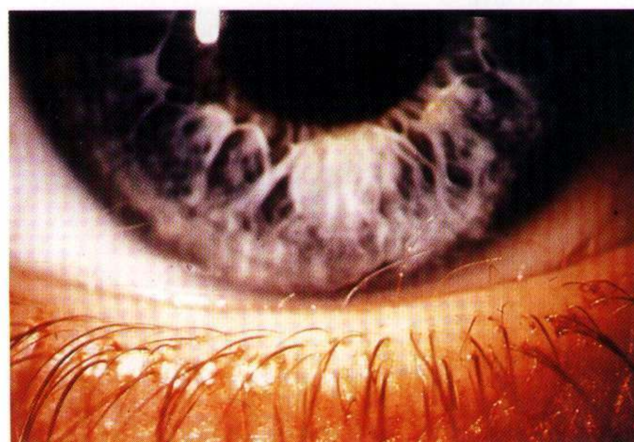


Fig. 1.9
Congenital distichiasis

c. The spot size is increased to 200 μm and the crater deepened to reach the follicle (Fig. 1.7).
d. About a dozen applications are required and most patients are cured by either one or two sessions.

5. **Surgery** involving full-thickness wedge resection (Fig. 1.8) or anterior lamellar excision may be useful for a localized crop of lashes resistant to other methods of treatment. More generalized trichiasis requires anterior lamellar reposition surgery.

Congenital distichiasis

Congenital distichiasis is a rare condition which may be sporadic or dominantly inherited. A minority of patients also manifest chronic lymphoedema, spinal arachnoid cysts and congenital heart defects (*lymphoedema–trichiasis syndrome*).

Signs

A partial or complete second row of lashes emerging at or slightly behind the meibomian gland orifices. The aberrant lashes tend to be thinner, shorter and less pigmented than normal cilia and are often directed posteriorly (Fig. 1.9).

Treatment

Treatment involves lamellar eyelid division and cryotherapy.

a. An incision is made along the grey line dividing the lid into anterior and posterior lamellae.
b. The posterior lamella and lash follicles are frozen with a double freeze–thaw cycle to –20°C (Fig. 1.10).
c. The lamellae are surgically re-apposed.

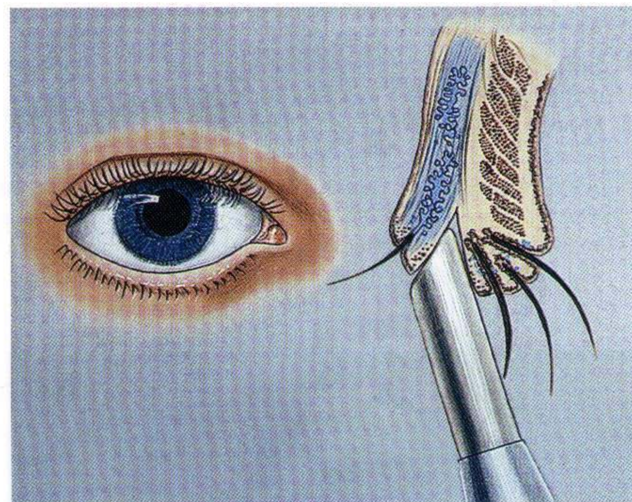


Fig. 1.10
Treatment of distichiasis by cryotherapy (Courtesy of Wilmer Institute)

Acquired distichiasis

Acquired distichiasis (metaplastic lashes) is caused by metaplasia and dedifferentiation of the meibomian glands to become hair follicles. The most important cause is late-stage cicatrizing conjunctivitis associated with chemical



Fig. 1.11
Acquired distichiasis (metaplastic lashes)

injury, Stevens–Johnson syndrome and ocular cicatricial pemphigoid.

1. **Signs**. Variable number of lashes which originate from meibomian gland orifices (Fig. 1.11).
2. **Treatment** of mild cases is as for trichiasis. Severe cases require lamellar eyelid division and cryotherapy to the posterior lamella.

Phthiriasis palpebrarum

The crab louse *Phthirus pubis* is adapted to living in pubic hair. An infested person may transfer the lice to another hairy area such as the chest, axilla or eyelid. Phthiriasis palpebrarum is an infestation of the lashes which typically affects children living in poor hygienic conditions and causes chronic irritation and itching.

Signs

The lice are anchored to the lashes by their thick claws (Fig. 1.12). The ova and their empty shells appear as oval, brownish, opalescent pearls adherent to the base of the cilia.



Fig. 1.12
Lice and nits clinging to the lashes in phthiriasis palpebrarum

Treatment

1. **Trimming of lashes** at their bases results in immediate mechanical removal of the lice and ova. It also destroys the hair-shaft habitat for louse survival and reproduction.
2. **Destruction** of lice and ova with topical application of yellow mercuric oxide 1% or anticholinesterase agents, laser or cryotherapy, if trimming is undesirable.
3. **Delousing** of the patient, other family members, clothing and bedding to prevent recurrences.

Poliosis

Poliosis is a premature localized whitening of hair which may involve the lashes and eyebrows (Fig. 1.13). The main causes are shown in Table 1.1.



Fig. 1.13
Poliosis of lashes

Table 1.1 Causes of poliosis

1. **Ocular**
   - chronic anterior blepharitis
   - sympathetic ophthalmitis
2. **Systemic**
   - Vogt–Koyangi–Harada syndrome
   - Waardenburg syndrome

Madarosis

Madarosis is a decrease in number or complete loss of lashes (Fig. 1.14). The main causes are shown in Table 1.2.

Hypertrichosis

Hypertrichosis is an excess number of lashes (polytrichosis) and/or abnormally long and luxuriant lashes (trichomegaly)



Fig. 1.14
Madarosis



Fig. 1.15
Hypertrichosis

Table 1.2 Causes of madarosis

1. **Local**
   - chronic anterior lid margin disease
   - infiltrating tumours
   - burns
   - radiotherapy or cryotherapy of lid tumours
2. **Skin disorders**
   - generalized alopecia
   - psoriasis
3. **Systemic disorders**
   - myxoedema
   - systemic lupus erythematosus
   - acquired syphilis
   - lepromatous leprosy
4. **Following removal**
   - iatrogenic for trichiasis
   - trichotillomania—psychiatric disorder of habitual hair removal

(Fig. 1.15). The condition may be congenital or drug-induced (e.g. phenytoin, cyclosporin and latanoprost).

Eyelash ptosis

Eyelash ptosis is a downward displacement of lashes (Fig. 1.16). The condition may be idiopathic or associated with the floppy eyelid syndrome, dermatochalasis with anterior lamellar slip or long-standing facial palsy.



Fig. 1.16
Eyelash ptosis

Allergic disorders

Acute allergic oedema

Acute allergic oedema is usually caused by insect bites, angioedema and urticaria, and occasionally drugs.



Fig. 1.17
Acute allergic oedema

1. **Signs**. Sudden onset of painless, pitting periorbital and lid oedema (Fig. 1.17).
2. **Treatment** with systemic antihistamines may be helpful.



Fig. 1.18
Contact dermatitis

Contact dermatitis

Contact dermatitis is caused by sensitivity to topical medication, either to the active component or the preservative. Common causes include neomycin, chloramphenicol and dorzolamide.

1. **Signs**. Lid swelling and erythema associated with tearing and itching. If the cause is not withdrawn the oedema lessens but the erythema persists and the skin becomes thickened and crusty (Fig. 1.18).
2. **Treatment** involves identification and removal of the cause and short-term application of a cream containing a mild steroid such as hydrocortisone 1%.

Atopic dermatitis

Atopic dermatitis (eczema) is a very common, idiopathic condition frequently associated with asthma and hay fever.



Fig. 1.19
Atopic dermatitis

Eyelid involvement is relatively infrequent but when present is invariably associated with generalized dermatitis.

1. **Signs**. Thickening, crusting and vertical fissuring of the lids associated with staphylococcal blepharitis and madarosis (Fig. 1.19).
2. **Treatment** is with emollients to hydrate the skin and the judicious use of mild topical steroids such as hydrocortisone 1%. It is also important to treat associated infection.
3. **Ocular associations**
   a. *Common*. Vernal disease in children and chronic keratoconjunctivitis in adults.
   b. *Uncommon*. Keratoconus, presenile cataract and retinal detachment.

Infections

Herpes zoster ophthalmicus

Herpes zoster ophthalmicus is a common, unilateral infection caused by varicella-zoster virus. It typically affects the elderly but may occur at an earlier age and be more severe in immunocompromised individuals.

Clinical features

1. **Presentation** is with pain in the distribution of the first division of the trigeminal nerve.
2. **Signs** (in chronological order)
   - A maculopapular rash on the forehead.
   - Progression through vesicles, pustules to crusting ulceration.



Fig. 1.20
Bilateral oedema due to unilateral herpes zoster ophthalmicus

- Periorbital oedema may spread to the other side, giving the erroneous impression that the condition is bilateral (Fig. 1.20).
- Ocular complications (*see* Chapter 5).

Treatment

1. **Systemic** treatment is for 7 days with valaciclovir 1 g t.i.d. or famciclovir 250 mg t.i.d. or 750 mg once daily.
2. **Topical** aciclovir or penciclovir cream, and a steroid–antibiotic combination such as Fucidin-H (hydrocortisone 1%, fucidic acid 2%) or Terra-Cortil (hydrocortisone 1%, oxytetracycline 3%). These should be used t.i.d. until the crusts have separated.

> *NB:* Talc and calamine lotion should be avoided.

Herpes simplex

Primary herpes simplex infection is an uncommon, unilateral condition which typically affects children. It may be particularly severe in patients with associated atopic dermatitis or immunodeficiency states.

1. **Signs**. Crops of small vesicles which rupture, crust and heal within a few days (Fig. 1.21). Uncommon complications include ipsilateral follicular conjunctivitis and keratitis.
2. **Treatment** is with aciclovir or penciclovir cream, taking care to avoid contact with the eyes.



Fig. 1.21
Vesicles due to primary herpes simplex infection

Impetigo

Impetigo is an uncommon, superficial skin infection caused by *Staph. aureus* or *Strep. pyogenes* which most frequently affects children. Involvement of the eyelids is usually associated with painful infection of the face.

1. **Signs**. Erythematous macules which rapidly develop into vesicles and bullae which produce golden-yellow crusts on rupturing (Fig. 1.22).



Fig. 1.22
Impetigo

2. **Treatment** is with topical antibiotics and oral flucloxacillin or erythromycin.

Erysipelas

Erysipelas (St Anthony fire) is an uncommon, acute subcutaneous spreading cellulitis usually caused by *Strep. pyogenes* through a site of minor skin trauma.

1. **Signs**. An expanding, well-defined, indurated, erythematous, subcutaneous plaque (Fig. 1.23). Primary lid involvement, when it occurs, is usually severe and may result in secondary contracture.
2. **Treatment** is with oral phenoxymethylpenicillin.



Fig. 1.23
Erysipelas due to minor skin trauma



Fig. 1.24
Gangrene due to necrotizing fasciitis

Necrotizing fasciitis

Necrotizing fasciitis is an extremely rare rapidly progressive necrosis initially involving subcutaneous soft tissues and later usually the skin which is usually caused by *Strep. pyogenes* and occasionally by *Staph. aureus*. The most frequent sites of involvement are the extremities, trunk and perineum, as well as postoperative wound sites. Unless treatment is early and appropriate, death may result. Periocular infection is rare and may be secondary to trauma or surgery.

1. **Signs**. Periorbital redness and oedema leading to formation of large bullae and black discoloration of skin due to gangrene secondary to underlying thrombosis (Fig. 1.24).
2. **Complications** include ophthalmic artery occlusion, lagophthalmos and disfigurement.
3. **Treatment** is with intravenous benzylpenicillin, debridement of necrotic tissue and reconstructive surgery.

Chronic marginal blepharitis

Pathogenesis

The pathogenesis of anterior blepharitis is unclear although both staphylococcal infection and seborrhoea play important roles (Table 1.3).

1. **Seborrhoeic blepharitis** is usually associated with seborrhoeic dermatitis which may involve the scalp, nasolabial folds, retroauricular areas and sternum. It has been postulated that excessive neutral lipids are broken down by *Corynebacterium acnes* into irritating fatty acids.
2. **Posterior blepharitis** is manifest as meibomian gland dysfunction (ocular rosacea) which may be associated with facial rosacea (*see* Chapter 20).

> *NB:* Because of the intimate relationship between the lids and ocular surface, chronic blepharitis may cause secondary changes in the conjunctiva and cornea.

Table 1.3 Classification of chronic blepharitis

1. **Anterior**
   - staphylococcal
   - seborrhoeic
   - mixed
2. **Posterior**
   - meibomian seborrhoea
   - meibomianitis
3. **Mixed anterior and posterior**

Anterior blepharitis

Clinical features

1. **Symptoms** include burning, grittiness, mild photophobia, and crusting and redness of the lid margins. These are usually worse in the mornings and are characterized by remissions and exacerbations. Surprisingly there is frequently little correlation between severity of symptoms and extent of clinical involvement.
2. **Signs**
   a. ***Staph. blepharitis*** is characterized by hyperaemia and telangiectasia of the anterior lid margins with hard scales mainly located around the bases of the lashes (collarettes) (Figs 1.25 and 1.26).
   b. ***Seborrhoeic blepharitis*** is characterized by hyperaemic and greasy anterior lid margins with sticking together of lashes (Fig. 1.27). The scales are soft and located anywhere on the lid margin and lashes (Fig. 1.28).
   c. ***Severe long-standing*** anterior blepharitis, particularly staphylococcal, may result in hypertrophy and scarring of the lid margin, madarosis (Fig. 1.29), trichiasis and poliosis (Fig. 1.30).

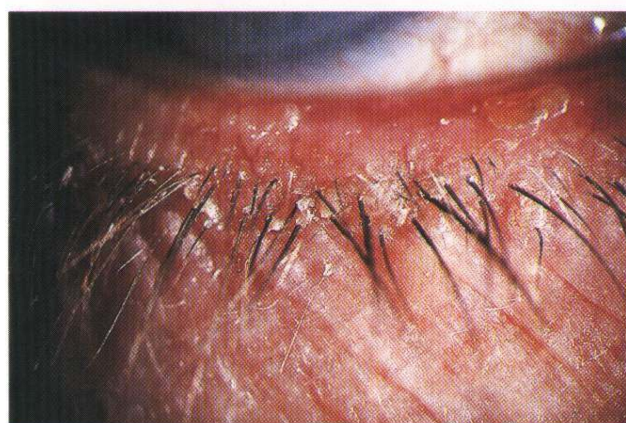
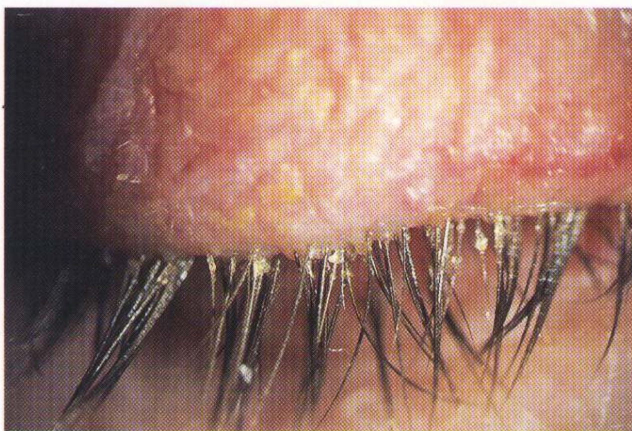


Fig. 1.25
Telangiectasia of the anterior lid margin and scales in staphylococcal blepharitis


Fig. 1.26
Hard scales in staphylococcal blepharitis

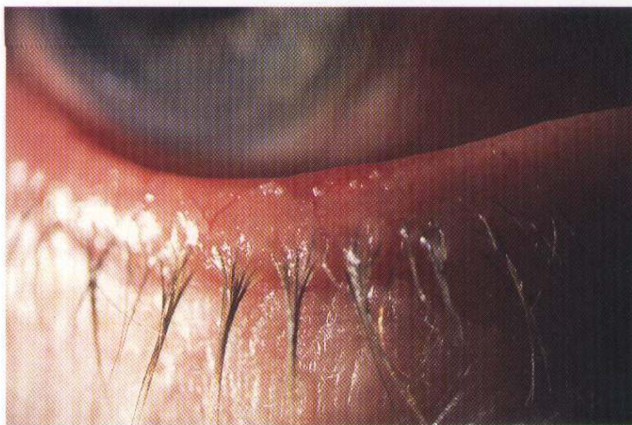
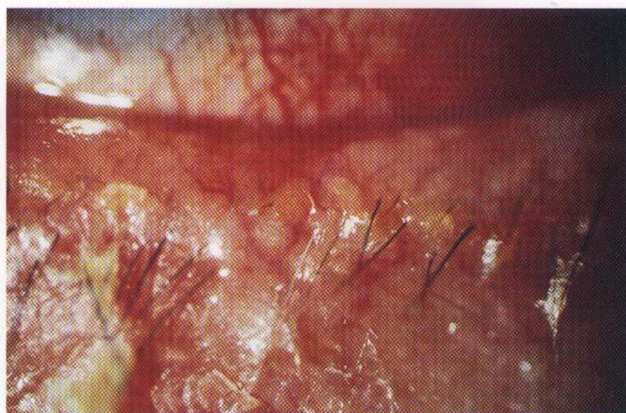
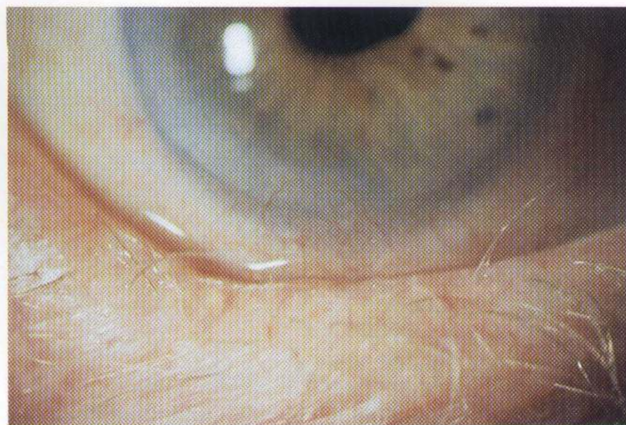

Fig. 1.27
Greasy lashes in seborrhoeic blepharitis


Fig. 1.28
Greasy lashes and scales in seborrhoeic blepharitis

3. Associations
- External hordeola (styes) may develop by spread of infection to the glands of Moll or Zeis.
- Tear film instability is present in 30–50% of cases.


Fig. 1.29
Notching, scarring and madarosis in long-standing staphylococcal blepharitis

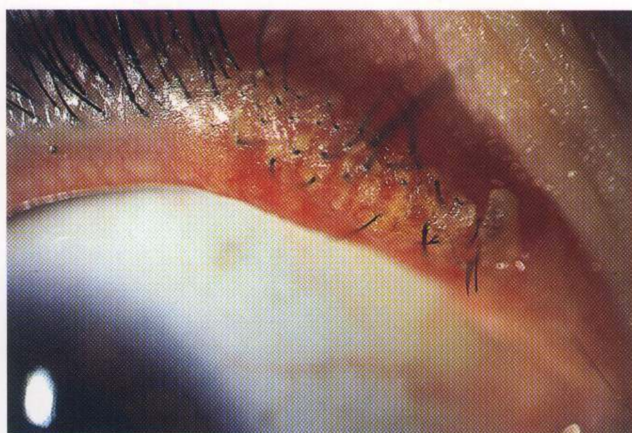

Fig. 1.30
Trichiasis and poliosis in long-standing staphylococcal blepharitis

- Hypersensitivity to staphylococcal exotoxins may result in papillary conjunctivitis, inferior corneal epithelial punctate erosions and marginal keratitis.

Treatment

This is tedious and patients should be advised that despite the lack of permanent cure control of symptoms is usually possible. In long-standing cases several weeks of intensive treatment may be necessary to achieve improvement.

1. **Lid hygiene** to remove crusts and toxic products involves scrubbing the lid margins daily with a commercially available lid scrub, a cotton bud dipped in a 25% solution of baby shampoo or a weak solution of sodium bicarbonate. Alternatively a face cloth or handkerchief can be used. It is also useful to scrub the eyelids with diluted shampoo when washing the hair. Gradually, lid hygiene can be performed less frequently as the condition is brought under control but must not be stopped or the blepharitis will recur.
2. **Antibiotic ointment** such as sodium fusidate (Fucidin) or chloramphenicol is used to treat acute folliculitis but is


Fig. 1.31
Sebaceous gland carcinoma causing localized loss of lashes and mimicking chronic blepharitis

of limited value in long-standing cases. Following lid hygiene the ointment should be rubbed onto the anterior lid margin with a cotton bud or clean finger.

3. **Weak topical steroids** such as fluorometholone administered short term q.i.d. are useful for secondary papillary conjunctivitis and marginal keratitis.
4. **Tear substitutes** are required for associated tear film instability. Unless this aspect of the disease is recognized and treated relief of symptoms will be incomplete.

Differential diagnosis

1. **Dry eye** can cause similar symptoms, but in contrast to blepharitis, ocular irritation is seldom severe in the morning and usually develops later in the day.
2. **Infiltrating lid tumours** should be suspected in patients with apparently asymmetrical or unilateral chronic blepharitis, particularly when associated with madarosis (Fig. 1.31).

Posterior blepharitis

Meibomian seborrhoea

This is characterized by excessive meibomian gland secretions. It is easy to miss because symptoms may be severe but clinical signs of blepharitis are mild.

1. **The meibomian gland orifices** are capped by small oil globules (Fig. 1.32). Pressure on the tarsus results in expression of copious amounts of meibomian oil.
2. **The tear film** is oily and foamy, and in severe cases froth accumulates on the lid margins or inner canthi (meibomian foam) (Fig. 1.33).

Meibomianitis

This is characterized by inflammation and obstruction of the meibomian glands.

1. **The posterior lid margin** shows hyperaemia, telangiectasia and obstruction of meibomian gland orifices (Figs 1.34 and 1.35). Long-standing cases are charac-

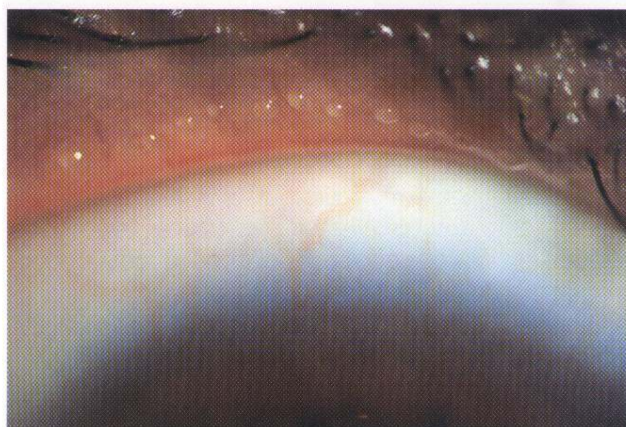

Fig. 1.32
Capping of meibomian gland orifices with oil in posterior blepharitis


Fig. 1.33
Foam in meibomian seborrhoea


Fig. 1.34
Diffuse inflammation around meibomian gland orifices in meibomianitis

terized by cystic dilatation of meibomian ducts, with thickening and notching of the lid margin (Fig. 1.36).

2. **Expressed meibomian gland secretions** in long-standing cases may be turbid or inspissated, appearing as toothpaste-like plaques (Fig. 1.37). In very severe cases no secretions can be expressed.

Complications

- Chalazion formation, which may be recurrent (Fig. 1.38).
- Tear film instability in about 30% of patients. This is probably the result of imbalance between the aqueous and lipid components of the tear film, allowing increased evaporation and dryness.
- Papillary conjunctivitis and inferior corneal epithelial erosions.

Treatment

1. **Systemic tetracyclines** are the mainstay of treatment but should not be used in children under the age of 12 years or in pregnant or breast-feeding women because they are deposited in growing bone and teeth (being bound to calcium), and may cause staining of teeth and dental hypoplasia. One of the following preparations may be used:
   - Tetracycline 250 mg q.i.d. for 1 week and then b.d. for 6–12 weeks.
   - Doxycycline 100 mg b.d. for 1 week and then daily for 6–12 weeks.
   - Minocycline 100 mg daily for 6–12 weeks.
2. **Erythromycin** or azithromycin may be used when tetracyclines are contraindicated, but their efficacy in posterior blepharitis is not well established.
3. **Other measures**
   - Lid hygiene, weak topical steroids and tear substitutes as for anterior blepharitis.
   - Warm compresses to melt solidified sebum and mechanical expression of the meibomian glands to reduce the amount of irritating lipids within the glands.
   - Topical sodium fusidate gel in patients with associated acne rosacea.



Fig. 1.35
Blocked meibomian gland orifices and scarring of the posterior lid margin in long-standing posterior blepharitis



Fig. 1.36
Obstruction of meibomian glands and telangiectasia of the posterior lid margin in long-standing posterior blepharitis



Fig. 1.37
Toothpaste-like plaques expressed from meibomian glands in posterior blepharitis

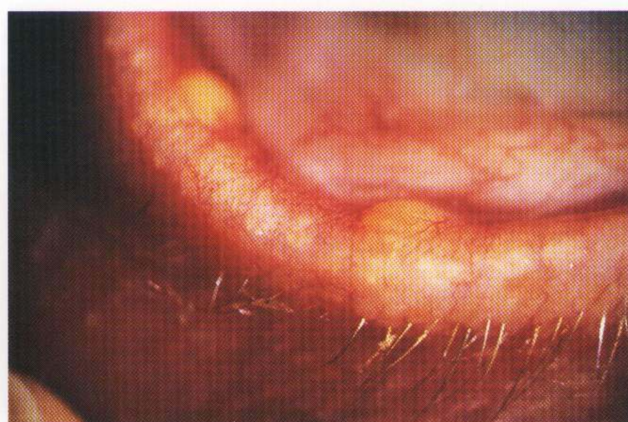


Fig. 1.38
Small chalazia associated with posterior blepharitis

Benign nodules and cysts

Chalazion

A chalazion (meibomian cyst) is a chronic, sterile, lipogranulomatous inflammatory lesion caused by blockage of meibomian gland orifices and stagnation of sebaceous secretions. Patients with acne rosacea or seborrhoeic dermatitis are at increased risk of chalazion formation, which may be multiple or recurrent.

Clinical features

1. **Presentation** is at any age with a gradually enlarging painless nodule. Occasionally an upper lid chalazion may press on the cornea, induce astigmatism and cause blurred vision.
2. **Signs**. A non-tender, roundish, firm lesion within the tarsal plate of variable size which may be multiple or bilateral (Fig. 1.39). Eversion of the lid may show an associated polypoidal granuloma if the lesion has ruptured through the tarsal conjunctiva (Fig. 1.40).



Fig. 1.39
Chalazion



Fig. 1.40
Conjunctival granuloma associated with a chalazion

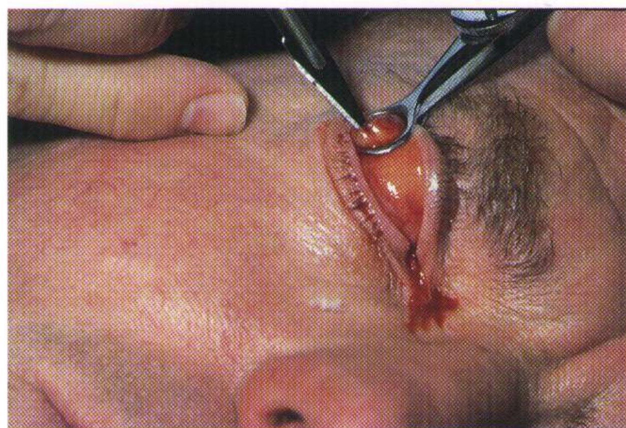


Fig. 1.41
Incision of chalazion

Treatment

Small chalazia may occasionally disappear spontaneously. Persistent lesions may be treated as follows:

1. **Surgery**. The eyelid is everted with a special clamp, the cyst is incised vertically and its contents curetted through the tarsal plate (Fig. 1.41). It is very important not to mistake a sebaceous gland carcinoma for a 'recurrent chalazion'. In doubtful cases, the lesion should be biopsied and examined histologically.
2. **Steroid injection** into the lesion. Between 0.1 and 0.2 ml triamcinolone diacetate aqueous suspension diluted with lignocaine (or equivalent) to a concentration of 5 mg/ml is injected through the conjunctiva with a 30-gauge needle. The success rate following one injection is about 80%. In unresponsive cases a second injection can be given 2 weeks later.
3. **Systemic tetracycline** may be required as prophylaxis in patients with recurrent chalazia, particularly if associated with acne rosacea or seborrhoeic dermatitis.

Internal hordeolum

An internal hordeolum is an abscess caused by an acute staphylococcal infection of a meibomian gland.

1. **Signs**. A tender, painful swelling within the tarsal plate (Fig. 1.42). The lesion may enlarge and then discharge either posteriorly through the conjunctiva or anteriorly through the skin.
2. **Treatment** by incision and curettage may be required if a residual nodule remains after the acute infection has subsided.

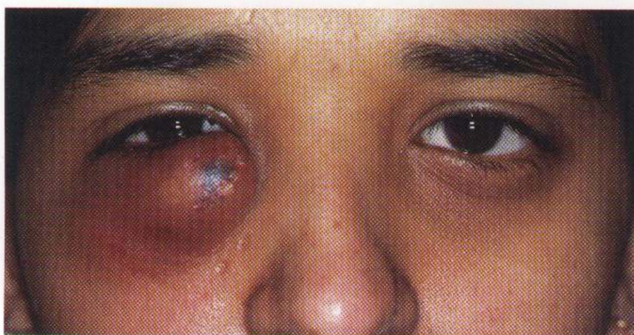


Fig. 1.42
Large internal hordeolum about to discharge

External hordeolum

An external hordeolum (stye) is an acute staphylococcal abscess of a lash follicle and its associated gland of Zeis or Moll which usually affects children.

1. **Signs**. A tender swelling in the lid margin pointing anteriorly through the skin (Fig. 1.43). Multiple lesions may be present and occasionally minute abscesses may involve the entire lid margin.
2. **Treatment** with hot compresses and epilation of the lash associated with the infected follicle may hasten resolution.

Molluscum contagiosum

Molluscum contagiosum is a skin infection caused by a pox-virus which typically affects otherwise healthy children. Immunocompromised patients, however, may have atypical multiple and frequently confluent lesions which are more resistant to treatment than in healthy individuals.

1. **Signs**. Pale, waxy, umbilicated nodule (Fig. 1.44).

> *NB:* Patients may occasionally present with a secondary ipsilateral chronic follicular conjunctivitis and unless the lid margin is examined carefully the causative molluscum lesion may be overlooked.



Fig. 1.43
External hordeolum (stye)



Fig. 1.44
Molluscum contagiosum

2. **Treatment** may not be necessary unless the lesion is very close to the lid margin. Treatment options include shave excision or destruction of the lesion by cauterization, cryotherapy or laser.

Xanthelasma

Xanthelasma is a common, frequently bilateral condition which is usually found in middle-aged and elderly patients or those with hyperlipidaemia.

1. **Signs**. Yellowish, subcutaneous plaques consisting of cholesterol and lipid which are usually located at the medial aspects of the eyelids (Fig. 1.45).
2. **Treatment** for cosmetic reasons is either by excision or, preferably, destruction with a carbon dioxide or argon laser.

Cyst of Moll

A cyst of Moll (apocrine sweat gland hidrocystoma) is a small, round, non-tender, translucent fluid-filled lesion on the anterior lid margin (Fig. 1.46).



Fig. 1.45
Xanthelasma

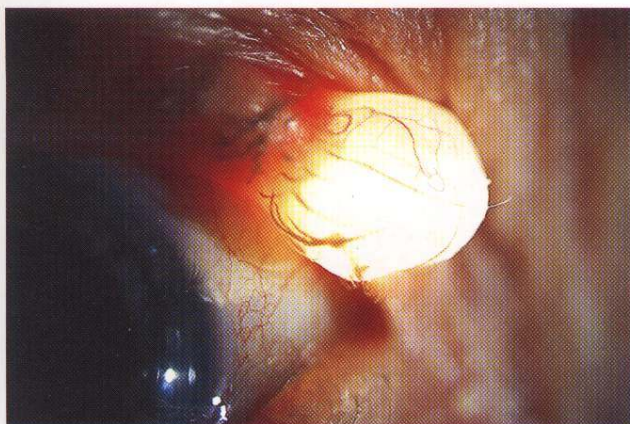
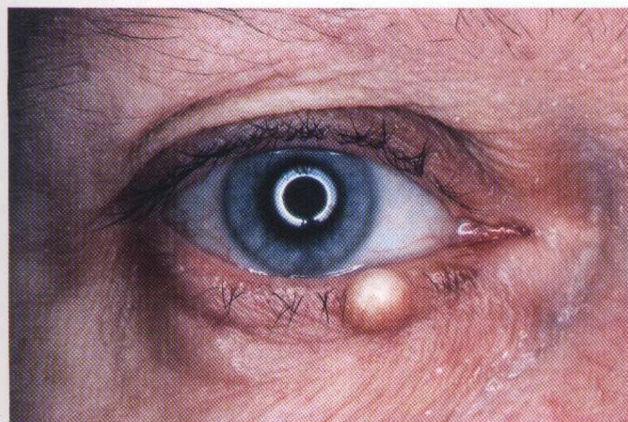

Fig. 1.46
Cyst of Moll


Fig. 1.47
Large eccrine sweat gland hidrocystoma at the outer canthus and a cyst of Moll on the upper lid


Fig. 1.48
Cyst of Zeis

Eccrine sweat gland hidrocystoma

An eccrine sweat gland hidrocystoma is less common but similar in appearance to a cyst of Moll, except that it is not confined to the lid margin (Fig. 1.47).

Cyst of Zeis

A cyst of Zeis contains oily secretions and is therefore less translucent than a cyst of Moll (Fig. 1.48).

Sebaceous cyst

A sebaceous cyst arises from an ordinary sebaceous gland and is characterized by a central punctum with retained cheesy secretions. It is rarely found on the eyelid although it may occur at the inner canthus (Fig. 1.49).

Milia

Milia are tiny, white, round, superficial cysts which tend to occur in crops (Fig. 1.50). They are derived from hair follicles or sebaceous glands.


Fig. 1.49
Sebaceous cysts


Fig. 1.50
Milia

Benign tumours

Viral wart

A viral wart (squamous cell papilloma) is the most common benign tumour of the eyelids which is usually found in adults.

1. **Signs**. A pedunculated or broad-based (sessile) lesion with a characteristic raspberry-like surface (Fig. 1.51).
2. **Treatment** involves excision or laser ablation.

Seborrhoeic keratosis

Seborrhoeic keratosis (basal cell papilloma) is a common, slow-growing condition found on the face and eyelids of elderly individuals.

1. **Signs**. A discrete, greasy, brown, flat, round or oval lesion with a friable verrucous surface and a 'stuck-on' appearance (Fig. 1.52). Occasionally the lesion may be pedunculated.
2. **Treatment** involves curettage of small flat lesions and excision of pedunculated lesions.

Actinic keratosis

Actinic (solar) keratosis is the most common pre-malignant skin lesion but rarely develops on the eyelids. It typically affects elderly, fair-skinned individuals who have been exposed to excessive sunlight and most frequently occurs on the forehead and backs of the hands.

1. **Signs**. A flat, scaly, hyperkeratotic lesion (Fig. 1.53). Occasionally the lesion is nodular or wart-like and may be associated with a cutaneous horn.
2. **Treatment** involves biopsy to confirm the diagnosis, followed by either excision or cryotherapy.

Cutaneous horn

A cutaneous horn is an uncommon lesion which is occasionally associated with underlying actinic keratosis or squamous cell carcinoma.

1. **Signs**. A hyperkeratotic lesion protruding through the skin (Fig. 1.54).
2. **Treatment** involves biopsy to determine the underlying pathology, followed by excision.

Pyogenic granuloma

A pyogenic granuloma is a fast-growing vascularized proliferation of granulomatous tissue which is usually antedated by surgery, trauma or infection, although some cases are idiopathic.

1. **Signs.** A pinkish, pedunculated or sessile mass which may bleed following relatively trivial trauma (Fig. 1.55).
2. **Treatment** is by excision.



Fig. 1.51
Pedunculated viral wart



Fig. 1.52
Seborrhoeic keratosis

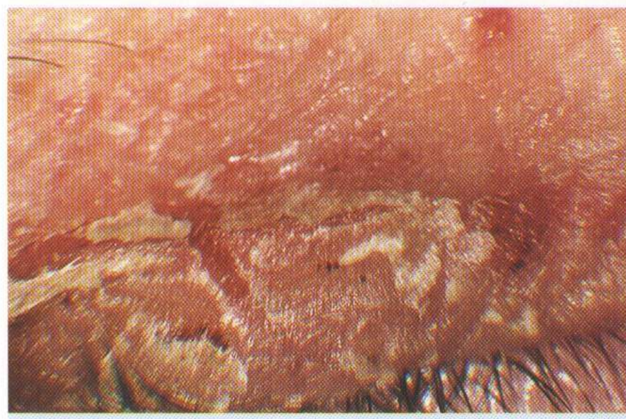


Fig. 1.53
Actinic keratosis


Fig. 1.54
Cutaneous horn


Fig. 1.55
Pyogenic granuloma

Melanocytic naevus

A melanocytic naevus is composed of atypical melanocytes (naevus cells). The clinical appearance, classification and potential for malignant transformation of naevi are determined by their histological location within the skin as follows:

1. **Intradermal naevus**, the most common, is usually elevated and frequently has a papillomatous configuration (Fig. 1.56). It may be non-pigmented or brown-black in colour. When located on the eyelid margin, lashes may grow through the lesion. Occasionally symmetrical lesions may be present on adjacent portions of the upper and lower lids ('kissing naevi') (Fig. 1.57). Histologically the naevus cells are confined to the dermis and have no malignant potential.
2. **Junctional naevus** is usually flat, well circumscribed and uniformly brown in colour (Fig. 1.58). The naevus cells are located at the junction of the epidermis and dermis and have a low potential for malignant transformation.
3. **Compound naevus** has both intradermal and junctional components and is usually brown. It has a low malignant potential which is related to the junctional component.


Fig. 1.56
Pigmented intradermal naevus


Fig. 1.57
'Kissing naevi' (Courtesy of S. Webber)


Fig. 1.58
Pigmented junctional naevus

Keratoacanthoma

Keratoacanthoma is an uncommon, benign but rapidly growing tumour which usually occurs in otherwise healthy fair-skinned individuals with a history of chronic sun

exposure. It is found more frequently than would be expected by chance in patients on immunosuppressive therapy following renal transplants. Keratoacanthoma may have a clinically similar appearance to squamous cell carcinoma, particularly in immunosuppressed individuals. Histopathologically keratoacanthoma is regarded as part of the spectrum of squamous cell carcinoma, into which it may evolve.

1. **Signs** (in chronological order)
   - A pink papule which may double or treble in size within a few days.
   - The lesion generally stops growing and remains static for 2–3 months, after which time it starts to involute spontaneously.
   - At the end of the growth phase the lesion is a firm, dome-shaped nodule (Fig. 1.59).
   - During the period of regression the central part of the lesion becomes hyperkeratotic and a keratin-filled crater may develop (Fig. 1.60).
   - Complete involution may take up to a year and usually leaves a residual ugly scar.
2. **Treatment** is by excision, especially if there is no sign of spontaneous involution.

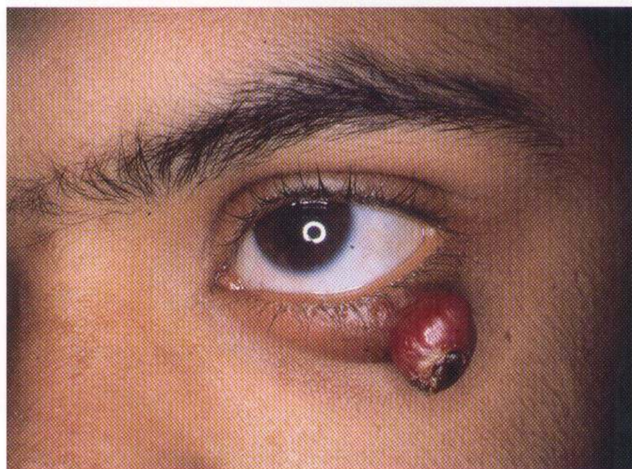


Fig. 1.59
Keratoacanthoma

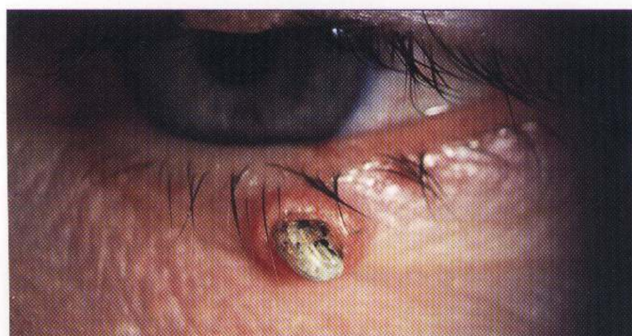


Fig. 1.60
Keratoacanthoma with a keratin-filled crater

Strawberry naevus

Strawberry naevus (capillary haemangioma) although rare is one of the most common tumours of infancy and presents shortly after birth. The female to male ratio is 3:1 and occasionally the tumour is familial. Eyelid angiomas have a predilection for the upper lid and may have orbital extensions. Occasionally the lesion may also involve the skin of the face (Fig. 1.61) and some patients have strawberry naevi on other parts of the body. It is important to be aware of the association between multiple cutaneous lesions and visceral haemangiomas.

Signs (in chronological order)

- A unilateral, raised, red lesion which blanches with pressure and may swell on crying (Fig. 1.62). A large tumour on the upper lid may cause mechanical ptosis (Fig. 1.63).



Fig. 1.61
Extensive capillary haemangioma



Fig. 1.62
Small capillary haemangioma

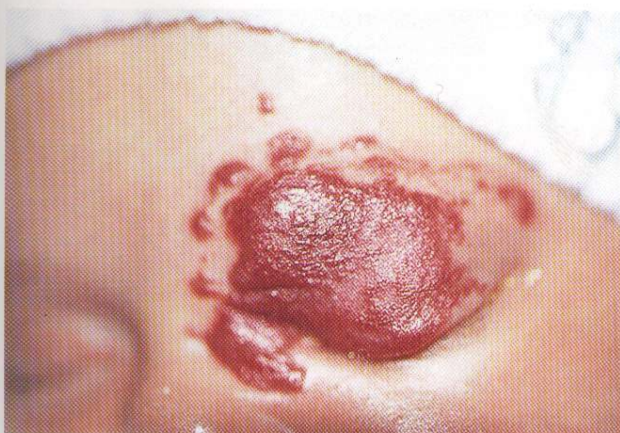


Fig. 1.63
Large capillary haemangioma

- The tumour usually grows quickly during the first year of life and stops growing during the second year.
- At about the age of 2 years the tumour begins to involute spontaneously with complete resolution occurring in 40% of patients by the age of 4 years and 70% by age 7.

Systemic associations

A small minority of patients with very large fast-growing naevi may have the following:

a. Kasabach–Merritt syndrome, which is characterized by thrombocytopenia, anaemia and low levels of coagulant factors.

b. Maffuci syndrome, which is characterized by skin haemangiomas, enchondromata of hands, feet and long bones as well as bowing of long bones.

Treatment

Treatment is indicated when vision is threatened by amblyopia as a result of anisometropia, ptosis or strabismus.

1. **Laser** treatment to close blood vessels in very early lesions.
2. **Injection** of steroid into the tumour is the most frequently used method. The injection contains a mixture, in equal parts, of triamcinolone acetonide 40 mg/ml and betamethasone 4 mg/ml. The tumour usually begins to regress within 2 weeks but, if necessary, second and third injections can be given after about 2 months. Potential complications are skin depigmentation, fat atrophy, eyelid necrosis and rarely occlusion of the central retinal artery.
3. **Systemic** steroids for extensive lesions, particularly if associated with visceral involvement.
4. **Subcutaneous** injection of interferon alpha-2b is a good option for the treatment of steroid-resistant, organ-interfering and/or life-threatening giant haemangiomas.
5. **Surgical resection** may be appropriate in selected cases.

Port-wine stain

A port-wine stain (naevus flammeus) is a rare congenital, subcutaneous cavernous haemangioma which most frequently occurs on the face. The lesion is usually unilateral and segmental but may be bilateral.

1. **Signs** (in chronological order)
   - A sharply demarcated, soft, pink patch which does not blanch with pressure (Fig. 1.64a).
   - With age, the lesion does not grow but darkens to red or purple (Fig. 1.64b).

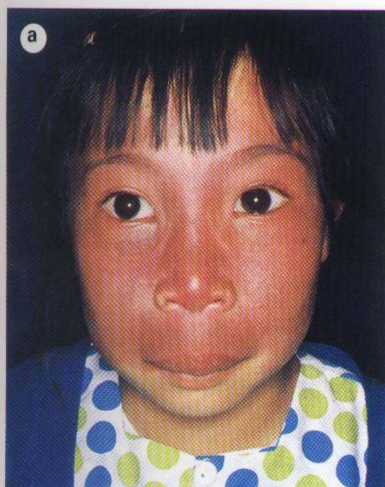






Fig. 1.64
Progression of skin changes in naevus flammeus. Darkening from red (**a**) to purple (**b**); (**c**) hypertrophy and nodularity

- The overlying skin may become hypertrophied, coarse, nodular and friable and may bleed or become infected (Fig. 1.64c).

2. **Associations** in patients with extensive lesions involving the first and second divisions of the trigeminal nerve are ipsilateral glaucoma (in about 30%), diffuse ipsilateral choroidal haemangioma and Sturge–Weber syndrome in 5% of cases (*see* Chapter 20).
3. **Treatment** with an erbium laser, if undertaken during early life, is effective in decreasing the amount of skin discoloration in relatively flat or mildly hypertrophic lesions.

Malignant tumours

Basal cell carcinoma

General features

Basal cell carcinoma (BCC) is the most common human malignancy, which most frequently affects elderly patients. The most important risk factors are fair skin, inability to tan and chronic exposure to sunlight. Ninety per cent of cases occur in the head and neck and, of these, about 10% involve the eyelid. BCC is by far the most common malignant eyelid tumour, accounting for 90% of all cases. It most frequently arises from the lower eyelid, followed in relative frequency by the medial canthus, upper eyelid and lateral canthus. The tumour is slow growing and locally invasive but non-metastasizing. Tumours located near the medial canthus are more prone to invade the orbit and sinuses, are more difficult to manage than those arising elsewhere and carry the greatest risk of recurrence. Tumours that recur following incomplete treatment tend to be more aggressive and difficult to manage.

Rare predisposing conditions

Young patients who suffer from one of these conditions may develop BCC:

1. **Xeroderma pigmentosum** is an autosomal recessive disease characterized by skin damage on exposure to natural sunlight which gives rise to progressive cutaneous pigmentation abnormalities. Affected patients have a rather bird-like facies and a great propensity to the development of skin BCC as well as squamous cell carcinoma and melanoma, which may be multiple (Fig. 1.65). Conjunctival malignancies have also been reported.
2. **Gorlin–Goltz syndrome** (naevoid basal cell carcinoma syndrome) is a rare autosomal dominant disorder characterized by extensive congenital deformities of the eye, face, bone and CNS. Many patients develop multiple, small BCC during the second decade of life. They are also predisposed to other malignancies including medulloblastoma, breast carcinoma and Hodgkin lymphoma.

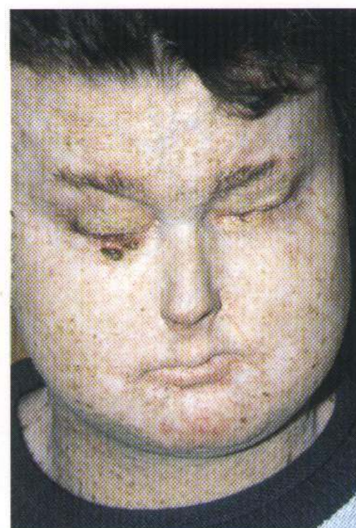


Fig. 1.65
Xeroderma pigmentosum and two eyelid basal cell carcinomas

Clinical types

1. **Nodulo-ulcerative** BCC starts as a shiny, firm, pearly nodule with small dilated blood vessels on its surface. Initially, growth is slow and it may take the tumour 1–2 years to reach a diameter of 0.5 cm (Fig. 1.66). If the tumour is not recognized and treated at an early stage, subsequent growth is faster and the lesion develops central ulceration (Fig. 1.67), raised rolled edges with dilated blood vessels over its lateral margins (rodent ulcer) (Fig. 1.68). With time it may erode a large portion of the eyelid (Fig. 1.69).
2. **Sclerosing** BCC (morphoeic) is less common and may be difficult to diagnose because it infiltrates laterally beneath the epidermis as an indurated plaque which may distort the eyelid (Fig. 1.70). The margins of the



Fig. 1.66
Small nodular basal cell carcinoma



Fig. 1.67
Small rodent ulcer

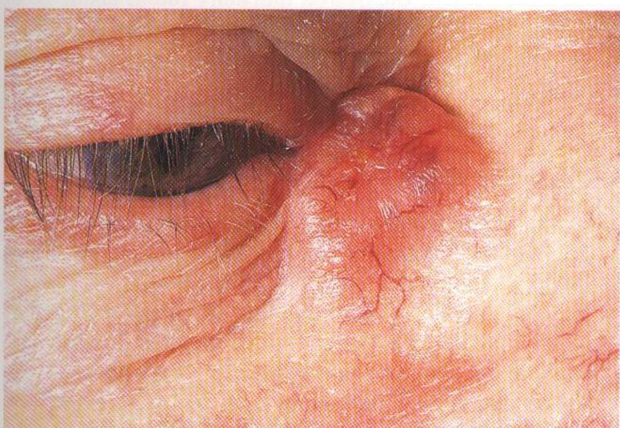


Fig. 1.68
Large rodent ulcer



Fig. 1.69
Erosion of the eyelid by a rodent ulcer

tumour may be impossible to delineate clinically and the lesion tends to be much more extensive on palpation than inspection. On cursory examination a sclerosing BCC may simulate a localized area of unilateral 'chronic blepharitis' (Fig. 1.71).



Fig. 1.70
Sclerosing basal cell carcinoma causing mechanical ectropion

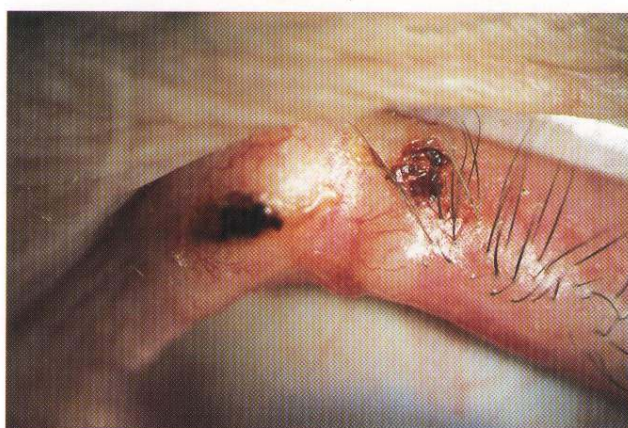


Fig. 1.71
Sclerosing basal cell carcinoma mimicking chronic blepharitis

Squamous cell carcinoma

General features

Squamous cell carcinoma (SCC) is a much less common, but potentially a more aggressive tumour than BCC, with eventual metastasis to regional lymph nodes. There may also be perineural spread to the intracranial cavity via the orbit. SCC accounts for 5–10% of eyelid malignancies and may arise *de novo* or from pre-existing actinic keratosis. Immunocompromised patients with AIDS or following renal transplants are at increased risk. The tumour has a predilection for the lower eyelid and the lid margin. It occurs most commonly in elderly individuals with a fair complexion and a history of chronic sun exposure and skin damage. The diagnosis of SCC may be difficult because certain benign-looking lesions such as keratoacanthoma may reveal histological evidence of invasive SCC at deeper levels of sectioning; conversely other malignant tumours, pre-cancerous lesions and benign tumours may mimic SCC. Clinically, SCC may be indistinguishable from a BCC but it does not usually manifest surface vascularization and grows more rapidly.

Clinical types

1. **Plaque-like** SCC is characterized by a roughened, scaly, erythematous, hyperkeratotic plaque which may arise at the site of pre-existing actinic keratosis (Fig. 1.72).
2. **Nodular** SCC is characterized by a hyperkeratotic nodule which may develop crusting erosions and fissures (Fig. 1.73).
3. **Ulcerating** SCC has a red base and sharply defined, indurated and everted borders (Fig. 1.74).



Fig. 1.72
Plaque-like squamous cell carcinoma (Courtesy of H. Frank)



Fig. 1.73
Nodular squamous cell carcinoma (Courtesy of H. Frank)



Fig. 1.74
Ulcerating squamous cell carcinoma (Courtesy of H. Frank)

Sebaceous gland carcinoma

General features

Sebaceous gland carcinoma (SGC) is a very rare slow-growing tumour which most frequently affects the elderly. It most frequently arises from the meibomian glands, although on occasion it may arise from the glands of Zeis or sebaceous glands. In contrast to BCC and SCC, the tumour occurs more commonly on the upper eyelid, where meibomian glands are more numerous. In about 5% of cases there is simultaneous involvement of both lids on one side, probably due to intra-epithelial spread or spontaneous development of multiple primaries. The clinical diagnosis of SGC is frequently difficult because, in its early stages, external signs of malignancy may be subtle so that the tumour may resemble a less aggressive lesion. As a result of frequent difficulties in diagnosis and delay in treatment, the overall mortality rate is about 10%. Bad prognostic features are upper lid involvement, tumour size of 10 mm or more and a duration of symptoms of over 6 months.

Clinical types

1. **Nodular meibomian gland carcinoma** presents with a discrete, hard nodule most commonly within the upper tarsal plate and may masquerade as a 'chalazion' (Fig. 1.75). It is therefore recommended that any chalazion of an unusual consistency should undergo full-thickness resection and histological examination. Unless treated, the nodule may become very large (Fig. 1.76).
2. **Spreading meibomian gland carcinoma** infiltrates into the dermis and causes a diffuse thickening of the lid margin (Fig. 1.77), similar to a sclerosing BCC, and may also invade the conjunctiva (Fig. 1.78). Pagetoid spread refers to extension of the tumour within the epithelium of the palpebral, forniceal or bulbar conjunctiva (Fig. 1.79). This may lead to the mistaken diagnosis of an inflammatory condition such as 'chronic conjunctivitis', 'superior limbic keratoconjunctivitis' or 'cicatricial pemphigoid'.
3. **Gland of Zeis carcinoma**, which is very rare, is characterized by a discrete, slow-growing, nodular or ulcerative lesion on the lid margin (Fig. 1.80).

Melanoma

Melanoma rarely develops on the eyelids but is potentially lethal. Although pigmentation is a hallmark of skin melanomas, half of lid melanomas are clinically non-pigmented and this may give rise to diagnostic difficulties.

Clinical types

1. **Superficial spreading melanoma** is characterized by a plaque with an irregular outline and variable pigmentation (Fig. 1.81).

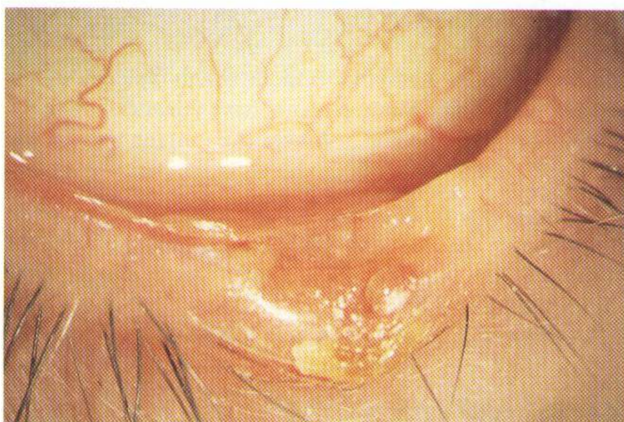

Fig. 1.75
Nodular meibomian gland carcinoma (Courtesy of H. Frank)


Fig. 1.76
Very large nodular meibomian gland carcinoma


Fig. 1.77
Spreading meibomian gland carcinoma


Fig. 1.78
Extensive involvement of the lid and adjacent conjunctiva by spreading meibomian gland carcinoma


Fig. 1.79
Involvement of the tarsal conjunctiva by spreading meibomian gland carcinoma (Courtesy of H. Frank)


Fig. 1.80
Ulcerating gland of Zeis carcinoma

2. **Nodular melanoma** is characterized by a blue-black nodule surrounded by normal skin (Fig. 1.82).
3. **Melanoma arising from lentigo maligna**. Lentigo maligna is a slowly expanding pigmented macule which typically affects the elderly and is also referred to as 'Hutchinson freckle'. Occasionally it is associated with the subsequent development of melanoma (Fig. 1.83).



Fig. 1.81
Superficial spreading melanoma



Fig. 1.82
Nodular melanoma

Kaposi sarcoma

Kaposi sarcoma is a vascular tumour which typically affects patients with AIDS. Many patients have advanced systemic disease although in some instances the tumour may be the sole manifestation of AIDS.

1. **Signs.** A small tumour is a pink, red-violet to brown lesion which may be mistaken for a haematoma or a naevus (Fig. 1.84). A large, rapidly growing tumour may ulcerate and bleed (Fig. 1.85).
2. **Treatment** is by radiotherapy or excision.

Merkel cell carcinoma

Merkel cell carcinoma is a fast-growing tumour arising from the dermis which typically affects the elderly. Its rarity may lead to difficulty in diagnosis and delay in treatment of this highly malignant and potentially lethal tumour. In fact, half the patients have metastatic spread at presentation.



Fig. 1.83
Extensive melanoma arising from lentigo maligna

1. **Signs**. A violaceous, well-demarcated nodule with intact overlying skin, most frequently involving the upper eyelid (Fig. 1.86).
2. **Treatment** is by excision which is frequently combined with chemotherapy.



Fig. 1.84
Kaposi sarcoma



Fig. 1.85
Large Kaposi sarcoma



Fig. 1.86
Merkel cell carcinoma (Courtesy of S. Webber)

Principles of treatment

Surgical excision

This should remove the entire tumour but at the same time preserve as much normal tissue as possible. Most small BCC can be cured by excision of the tumour together with a 4 mm margin of tissue which looks clinically normal. More radical surgical excision is required for large BCC and aggressive tumours such as SCC and SGC. Frozen section control by either a standard method or micrographic surgery can further improve the success rate.

1. **Standard frozen section** involves histological examination of the margins of the excised specimen at the time of surgery to ensure that they are tumour-free. If no tumour cells are detected, the eyelid is reconstructed; if some are present in a particular area, further excision is performed until the specimen is tumour-free.
2. **Moh micrographic surgery** involves excision with serial horizontal frozen sections from the undersurface of the tumour. The sections are then colour coded or mapped to identify any remaining areas of tumour. Although time-consuming, this maximizes the chances of total tumour excision with minimal sacrifice of normal tissue. This is a particularly useful technique for tumours that grow diffusely and have indefinite margins with finger-like extensions, such as sclerosing BCC, SGC, recurrent tumours and those involving the medial or lateral canthi.

Technique of reconstruction

This depends on the extent of full-thickness horizontal resection. It is important to reconstruct both anterior and posterior lamellae. If one of the lamellae has been sacrificed during excision of the tumour, it must be reconstructed with similar tissue. The technique depends on the size of the defect and laxity of the lid.

1. **Small defects** involving less than one-third of the eyelid can usually be closed directly, provided the surrounding tissue is sufficiently elastic to allow approximation of the cut edges. If necessary, a lateral cantholysis can be fashioned to mobilize additional tissue if the defect cannot be reapproximated (Fig. 1.87).
2. **Moderate-size defects** involving less than half of the eyelid may require a Tenzel semicircular flap for closure (Fig. 1.88).
3. **Large defects** involving over half of the eyelid may be closed by one of the following techniques:
   a. ***Mustarde cheek rotation flap*** is used for a lower eyelid defect (Fig. 1.89). The posterior lamella is reconstructed with nasal septum cartilage and mucosa or full-thickness buccal mucous membrane or a Hughes flap.
   b. ***Eyelid-sharing procedures*** may also be used, although care should be taken not to compromise upper lid function when reconstructing the lower lid. Figure 1.90 shows reconstruction of the entire lower eyelid following excision of an infiltrating basal cell carcinoma.
   c. ***Glabellar or rhomboid flaps*** may be required for defects involving the medial canthus and medial aspects of the upper eyelid (Fig. 1.91).

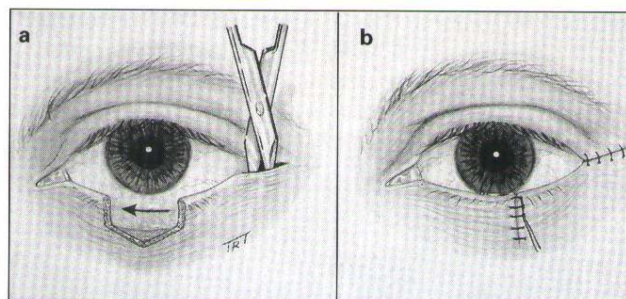


Fig. 1.87
(a) Lateral cantholysis; **(b)** to enhance closure of a small eyelid defect

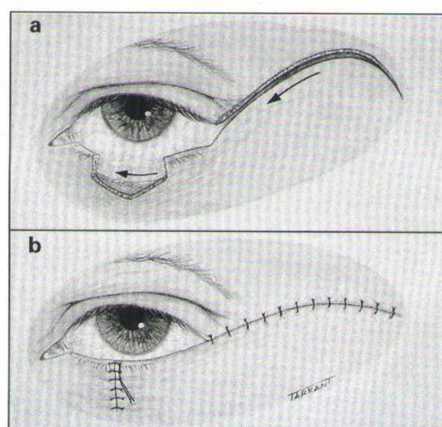


Fig. 1.88
(a) Large lid defect; **(b)** Tenzel flap

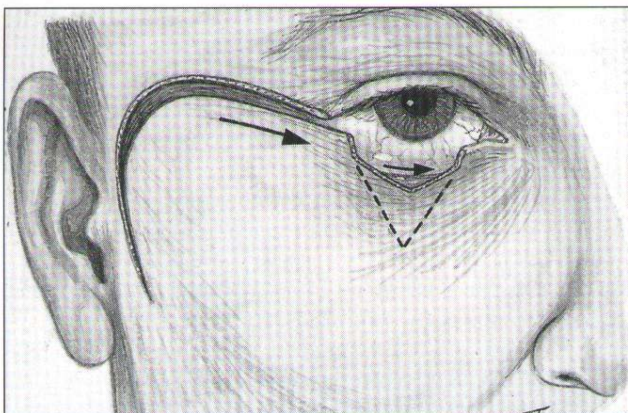


Fig. 1.89
Closure of a large inferior eyelid defect by a Mustarde cheek rotation flap

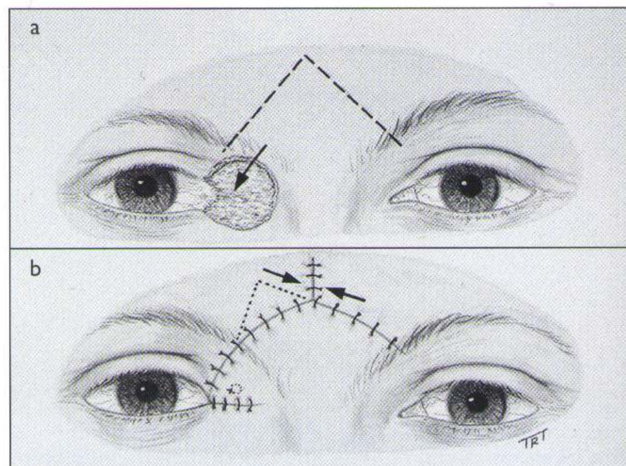


Fig. 1.91
Glabellar flap procedure following excision of a tumour at the medial canthus

Radiotherapy

1. Indications
- Small nodulo-ulcerative BCCs which do not involve the medial canthal area in patients who are either unsuitable for or refuse surgery.
- Kaposi sarcoma because it is radiosensitive.

2. Contraindications
- Medial canthal BCC because radiotherapy would damage the canaliculi and result in epiphora.
- Upper eyelid tumours because subsequent keratinization results in a chronically uncomfortable eye.
- Aggressive tumours such as sclerosing BCC, SCC and SGC.

3. Potential complications
- Skin damage and madarosis (Fig. 1.92).
- Nasolacrimal duct stenosis following irradiation to the medial canthal area.

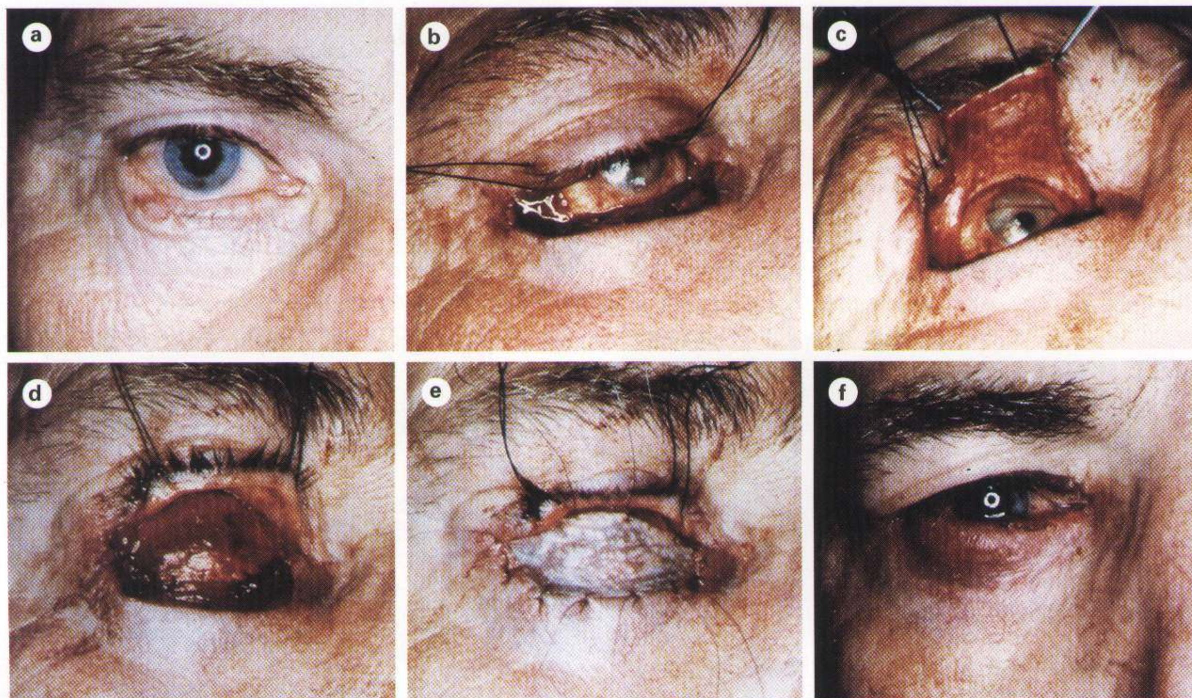


Fig. 1.90
Eyelid-sharing procedure. (**a**) Preoperative appearance of extensive sclerosing basal cell carcinoma; (**b**) appearance following total excision; (**c** and **d**) fashioning of a tarso-conjunctival flap to fill the defect in the posterior lamella; (**e**) reconstruction of the anterior lamella with a skin graft; (**f**) final appearance


Fig. 1.92
Scarring and madarosis induced by radiotherapy for a basal cell carcinoma

- Conjunctival keratinization, dry eye, keratopathy and cataract.
- Retinopathy and optic neuropathy.

> *NB:* Many of these complications can be avoided if the globe is protected by a special eyeshield during irradiation. The recurrence rate is higher than after surgery, and radiotherapy does not allow histological confirmation of tumour eradication. Recurrences following radiotherapy are difficult to treat surgically because of the poor healing properties of irradiated tissue.

Cryotherapy

This does not damage the canalicular system.

1. **Indications.** Small superficial BCC.
2. **Contraindications** are similar to those of radiotherapy, although cryotherapy may be a useful adjunct to surgery in patients with epibulbar pagetoid extensions of SGC, thus sparing the patient an exenteration.
3. **Potential complications** are skin depigmentation, madarosis and conjunctival overgrowth.

Laser microsurgery

This is a relatively new method of treating well-circumscribed BCC of the lid margins without conjunctival extension. The carbon dioxide laser acts as a scalpel to excise the tumour followed by vaporization of the deep and lateral resected margins. The wound is then left to heal by secondary intention.

Ectropion

Involutional ectropion

Involutional (age-related) ectropion affects the lower lid of elderly patients. It results in epiphora and in long-standing cases the tarsal conjunctiva may become chronically inflamed, thickened and keratinized.

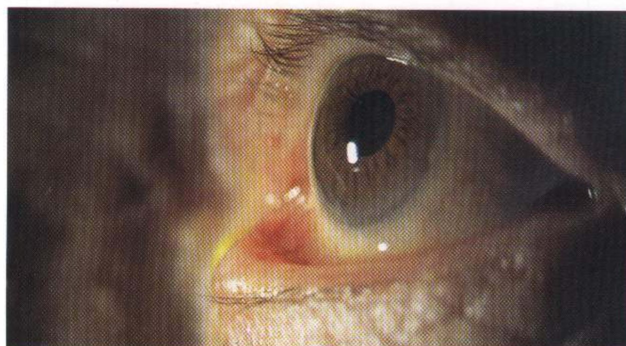
Pathogenesis

1. **Horizontal lid laxity** is demonstrated by pulling the central part of the lid 8 mm or more from the globe and its failure to snap back to its normal position on release without the patient first blinking (Fig. 1.93).
2. **Medial canthal tendon laxity** is demonstrated by pulling the lower lid laterally and observing the position of the inferior punctum (Fig. 1.94a). If the lid is normal the punctum should not be displaced more than 1–2 mm. If laxity is mild the punctum reaches the limbus, and if severe it reaches the pupil.
3. **Lateral canthal tendon laxity** is characterized by a rounded appearance of the lateral canthus and the ability to pull the lower lid medially more than 2 mm (Fig. 1.94b).

Treatment

The chosen method depends on the following: (a) extent of ectropion (i.e. predominantly medial or generalized), (b) extent of horizontal lid laxity, (c) severity of canthal tendon laxity and (d) degree of excess skin.

1. **Medial ectropion** (Fig. 1.95a) is treated by the Lazy-T procedure, which involves excision of a tarsoconjunctival diamond (medial spindle) about 4 mm high and 8 mm long, parallel with and inferior to the canaliculus and punctum, together with excision of a pentagon of full-thickness lid lateral to the punctum (Fig. 1.95b).
2. **Generalized ectropion** (Fig. 1.96a) is treated by horizontal lid shortening in which a pentagon of full-thickness lid is excised where the ectropion is most marked (Fig. 1.96b). Significant medial canthal tendon laxity should also be corrected.
3. **Marked generalized ectropion with excess skin** (Fig. 1.97a) is treated with the Kuhnt–Szymanowski procedure, in which a pentagon of full-thickness lid is excised laterally together with a lateral triangle of redundant skin (Fig. 1.97b). Significant medial canthal tendon laxity should also be corrected.


Fig. 1.93
Involutional ectropion

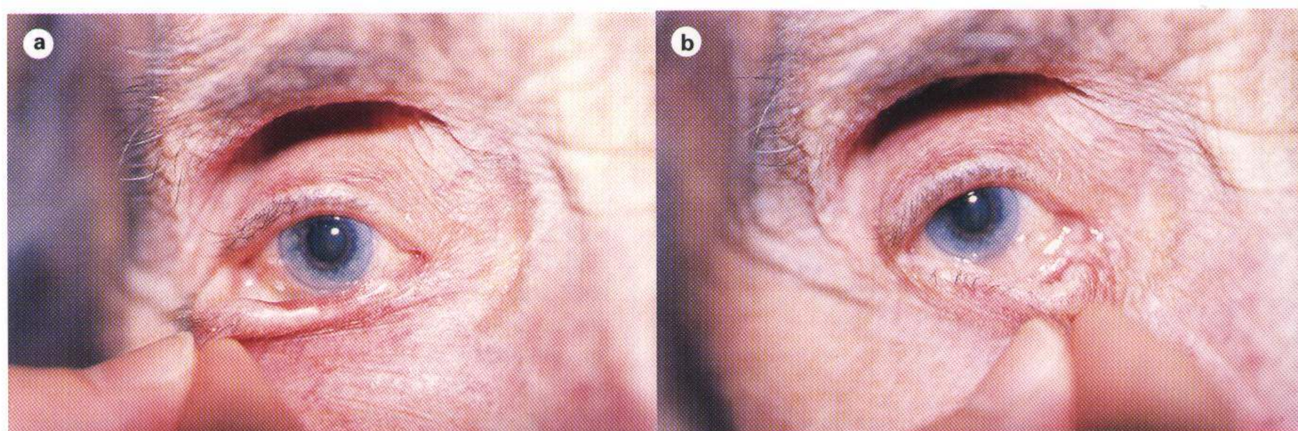


Fig. 1.94
Involutional ectropion. (**a**) Medial canthal tendon laxity; (**b**) lateral canthal tendon laxity

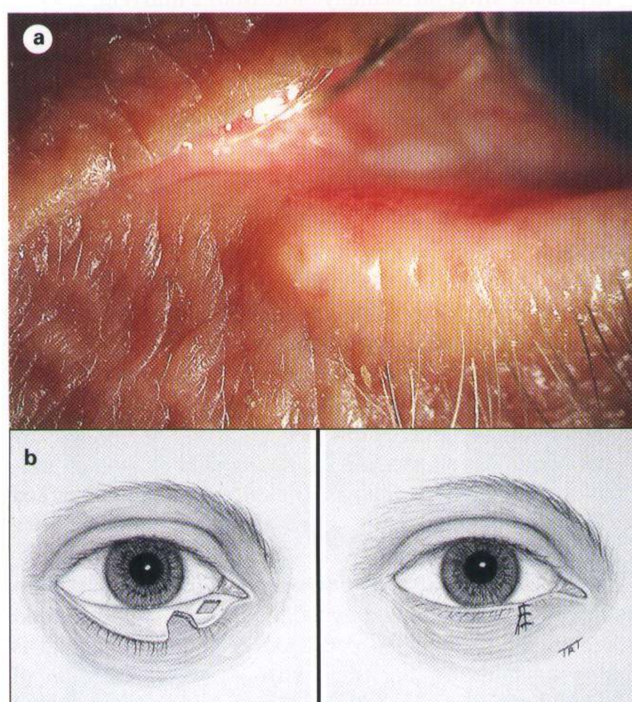


Fig. 1.95
(**a**) Medial ectropion; (**b**) lazy-T procedure

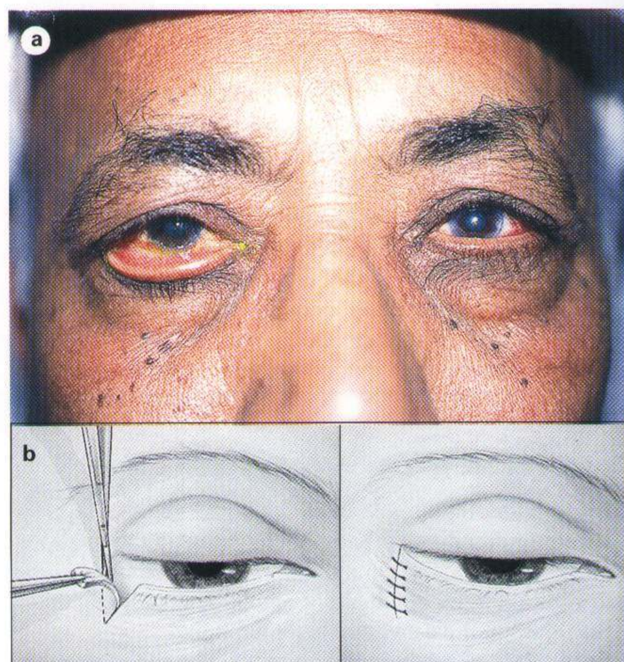


Fig. 1.96
(**a**) Generalized ectropion without excess skin; (**b**) horizontal lid shortening

Cicatricial ectropion

Cicatricial ectropion is caused by scarring or contracture of the skin and underlying tissues which pulls the eyelid away from the globe (Fig. 1.98). If the skin is pushed over the orbital margin with a finger the ectropion will be relieved and the lids will close. Opening the mouth tends to accentuate the ectropion. Depending on the cause, both lids may be involved and the defect may be local (e.g. trauma) or general (e.g. burns, dermatitis, ichthyosis).

1. **Mild localized** cases are treated by excision of the offending scar tissue combined with a procedure that lengthens vertical skin deficiency such as 'Z'-plasty (Fig. 1.99).
2. **Severe generalized** cases require transposition flaps or free skin grafts. Sources of skin include upper lids, as well as posterior auricular, preauricular and supraclavicular areas.

Paralytic ectropion

Paralytic ectropion is caused by an ipsilateral facial nerve palsy and is associated with retraction of the upper and lower lids and brow ptosis. The latter may cause narrowing of the palpebral aperture.

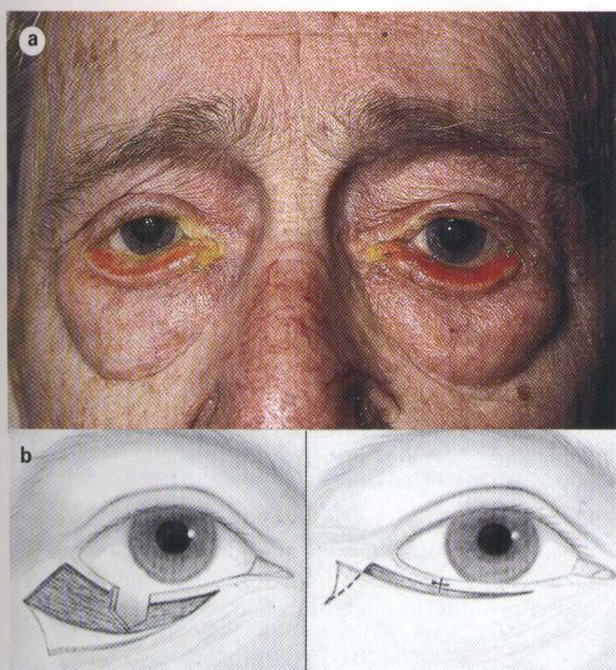


Fig. 1.97
(a) Generalized ectropion with excess skin;
(b) Kuhnt–Szymanowski procedure



Fig. 1.98
Cicatricial ectropion due to trauma

Potential complications

1. **Exposure keratopathy** is caused by a combination of lagophthalmos (Fig. 1.100) and inadequate resurfacing of the tear film over the cornea by the lids.
2. **Epiphora** is caused by malposition of the inferior lacrimal punctum, failure of the lacrimal pump mechanism and an increase in tear production resulting from corneal exposure.

Temporary treatment

This is aimed at protecting the cornea in anticipation of spontaneous recovery of facial nerve function.

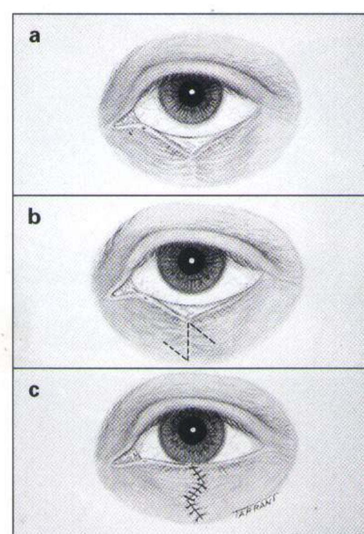


Fig. 1.99
Vertical lid-lengthening ('Z'-plasty)



Fig. 1.100
Bilateral paralytic ectropion and lagophthalmos due to facial nerve palsies

1. **Lubrication** with tear substitutes during the day and instillation of ointment and taping shut of the lids during sleep are usually adequate in mild cases.
2. **Temporary tarsorrhaphy**, a procedure in which the lateral aspect of the upper and lower lids are sutured together (Fig. 1.101), may be necessary in patients with a poor Bell phenomenon, in which the cornea remains exposed when the patient attempts to blink.

Permanent treatment

This should be considered when there has been no improvement of a Bell palsy after 3 months or when there is permanent damage to the facial nerve, as may occur following removal of an acoustic neuroma. Treatment is aimed at reducing horizontal and vertical dimensions of the palpebral aperture by one of the following procedures:

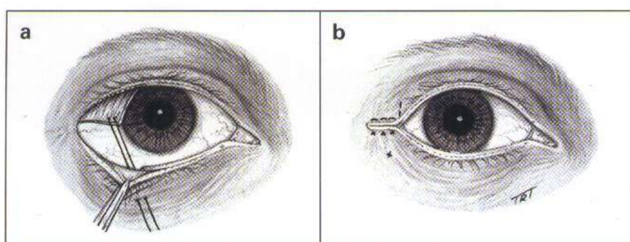


Fig. 1.101
Lateral tarsorrhaphy

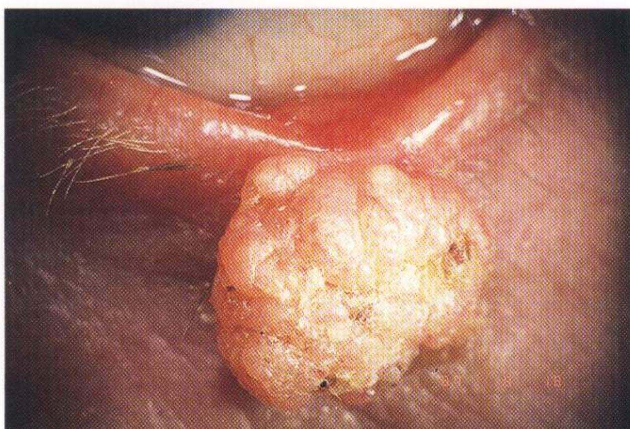


Fig. 1.102
Mechanical ectropion due to a tumour

1. **Medial canthoplasty** may be performed if the medial canthal tendon is intact. The eyelids are sutured together medial to the lacrimal puncta so that the puncta become inverted and the fissure between the inner canthus and puncta is shortened.
2. **Medial wedge resection** with attachment of tarsus to the posterior lacrimal crest is used to correct medial ectropion associated with medial canthal laxity.
3. **Lateral canthal sling** may be used to correct residual ectropion and raise the lateral canthus.

Mechanical ectropion

Mechanical ectropion is caused by tumours on or near the lid margin which mechanically evert the lid (Fig. 1.102). Treatment involves removal of the cause, if possible, and correction of significant horizontal lid laxity.

Entropion

Involutional entropion

Involutional (age-related) entropion affects mainly the lower lid because the upper has a broader tarsus and is more stable. The constant rubbing of the lashes on the cornea in patients with long-standing entropion (pseudo-trichiasis) may cause irritation, corneal punctate epithelial erosions and, in severe cases, ulceration and pannus formation (Fig. 1.103).

Pathogenesis

This involves age-related degeneration of elastic and fibrous tissues within the eyelid resulting in the following (Fig. 1.104):

1. **Horizontal lid laxity** caused by stretching of the canthal tendons and tarsal plate.
2. **Vertical lid instability** caused by attenuation, dehiscence or disinsertion of the lower lid retractors. Weakness of the latter is recognized by decreased excursion of the lower lid in downgaze.
3. **Over-riding** of the pretarsal by the preseptal orbicularis during lid closure tends to move the lower border of the tarsus anteriorly, away from the globe, and the upper border towards the globe, thus tipping the lid inwards.

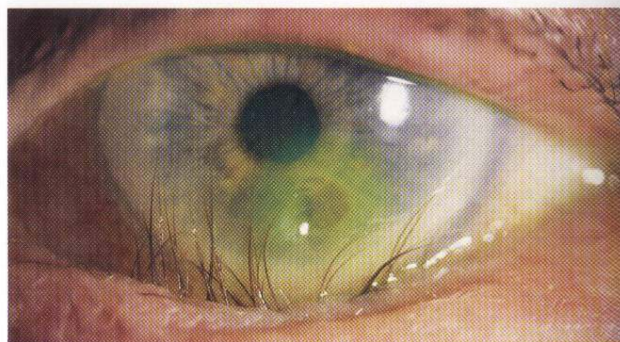


Fig. 1.103
Corneal ulceration due to pseudo-trichiasis associated with involutional entropion

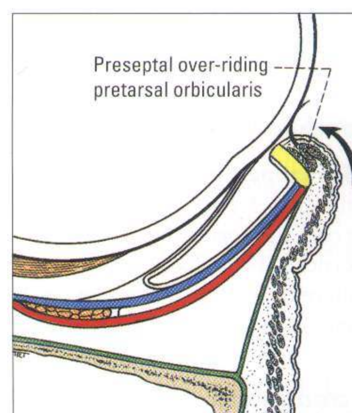


Fig. 1.104
Pathogenesis of involutional entropion

Treatment

Temporary treatment is with lubricants, taping, orbicularis chemodenervation with botulinum toxin injection or soft bandage contact lenses. Surgical treatment is determined principally by the severity of horizontal lid laxity.

1. **Horizontal lid laxity absent**
   a. *Transverse everting sutures* prevent over-riding of the preseptal orbicularis and provide temporary correction lasting several months (Fig. 1.105).
   b. *Weis procedure* gives a lasting correction (Fig. 1.106). It consists of full-thickness horizontal lid-splitting and insertion of everting sutures. The scar creates a barrier between the preseptal and pretarsal orbicularis, and the everting suture transfers the pull of the lower lid retractors from the tarsus to the skin and orbicularis.
   c. *Jones procedure* can be performed as primary treatment but is frequently reserved for recurrences. It tightens the lower lid retractors, thus increasing their pull, and creates a barrier between the preseptal and pretarsal orbicularis (Fig. 1.107).
2. **Horizontal lid laxity present**. This requires transverse lid splitting, inserting of everting sutures and horizontal lid shortening (Quickert procedure). Significant canthal tendon laxity should also be corrected.

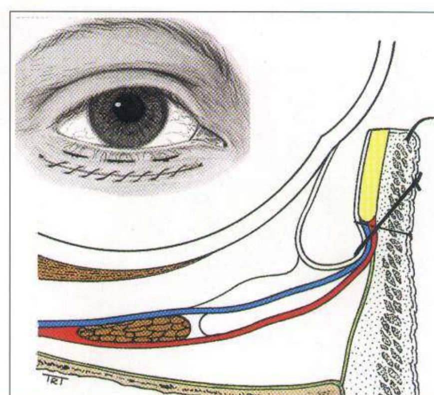


Fig. 1.106
Weis procedure

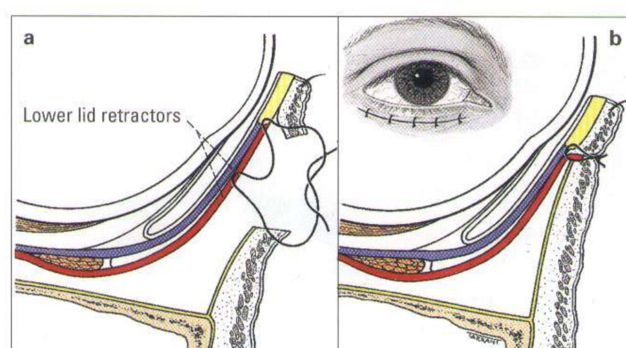


Fig. 1.107
Jones procedure

Cicatricial entropion

Cicatricial entropion is caused by severe scarring of the palpebral conjunctiva, which pulls the upper or lower lid margin towards the globe (Fig. 1.108). Causes include cicatrizing conjunctivitis, trachoma, trauma and chemical injuries.

1. **Medical** treatment is aimed at keeping the lashes away from the cornea by bandage contact lenses.
2. **Surgical** treatment of mild cases is by transverse tarsotomy (tarsal fracture) with anterior rotation of the lid margin. Treatment of severe cases is difficult and is directed at replacing deficient or keratinized conjunctiva and replacing the scarred and contracted tarsus with composite grafts.

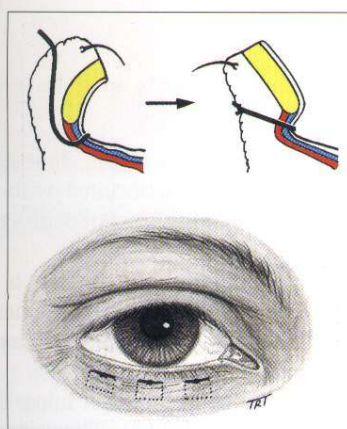


Fig. 1.105
Transverse lid-everting sutures

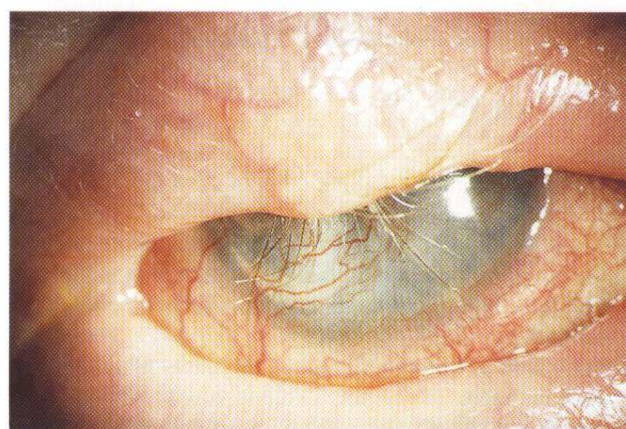


Fig. 1.108
Cicatricial upper lid entropion resulting in corneal scarring and vascularization



Fig. 1.109
Congenital lower lid entropion

Congenital entropion

Upper lid entropion

This is usually secondary to mechanical effects of microphthalmos which cause variable degrees of upper lid inversion.

Lower lid entropion

This is caused by improper development of the inferior retractor aponeurosis.

1. **Signs**. Inturning of the entire lower eyelid and lashes with absence of the lower lid crease (Fig. 1.109), which should not be confused with epiblepharon.
2. **Treatment** involves the excision of a strip of skin and muscle, and fixation of the skin crease to the tarsal plate (Hotz procedure).

Ptosis

Classification

Ptosis is an abnormally low position of the upper lid which may be congenital or acquired. An anatomical classification is shown in Table 1.4.

1. **Neurogenic ptosis** is caused by an innervational defect such as third nerve and oculosympathetic palsy (*see* Chapter 18).
2. **Myogenic ptosis** is caused by a myopathy of the levator muscle itself, or by impairment of transmission of impulses at the neuromuscular junction (neuromyopathic). Acquired myogenic ptosis occurs in myasthenia gravis, myotonic dystrophy (*see* Chapter 20) and ocular myopathies (*see* Chapter 18).

Table 1.4 Classification of ptosis

1. **Neurogenic**
   - third nerve palsy
   - Horner syndrome
   - Marcus Gunn jaw-winking syndrome
   - third nerve misdirection
2. **Myogenic**
   - myasthenia gravis
   - myotonic dystrophy
   - ocular myopathy
   - simple congenital
   - blepharophimosis syndrome
3. **Aponeurotic**
   - involutional
   - postoperative
4. **Mechanical**
   - dermatochalasis
   - tumours
   - oedema
   - anterior orbital lesions
   - scarring

3. **Aponeurotic** ptosis is caused by a defect in the levator aponeurosis.
4. **Mechanical** ptosis is caused by gravitational effect of a mass or scarring.

Clinical evaluation

History

The age at onset of ptosis and its duration will usually distinguish congenital from acquired cases. If the history is ambiguous, old photographs may be helpful. It is also important to enquire about symptoms of possible underlying systemic disease, such as associated diplopia, variability of ptosis during the day and excessive fatigue.

Pseudoptosis

This false impression of ptosis may be caused by the following:

1. **Lack of support** of the lids by the globe may be due to an orbital volume deficit associated with an artificial eye, microphthalmos, phthisis bulbi or enophthalmos (Fig. 1.110).
2. **Contralateral lid retraction**, which is detected by comparing the levels of the upper lids, remembering that the margin of the upper lid normally covers the superior 2 mm of the cornea (Fig. 1.111).
3. **Ipsilateral hypotropia**, because the upper lid follows the globe downwards (Fig. 1.112). The pseudoptosis will disappear when the hypotropic eye assumes fixation on covering the normal eye.



Fig. 1.110
Right pseudoptosis due to an artificial eye

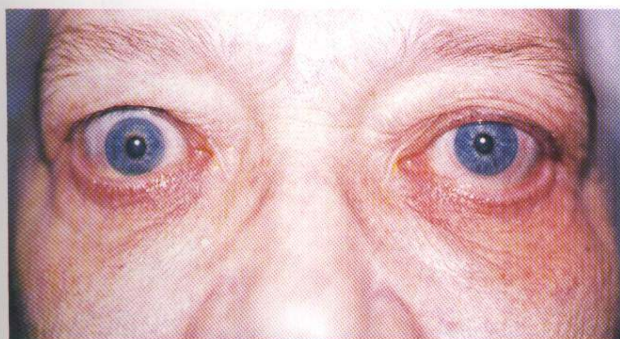


Fig. 1.111
Left pseudoptosis due to retraction of the right upper lid



Fig. 1.112
Left pseudoptosis due to ipsilateral hypotropia

4. **Brow ptosis** due to excessive skin on the brow or seventh nerve palsy which is diagnosed by manually elevating the eyebrow (Fig. 1.113).
5. **Dermatochalasis** in which excessive skin on the upper lids may cause both a pseudo- and a real ptosis (Fig. 1.114).



Fig. 1.113
Brow ptosis



Fig. 1.114
Pseudoptosis due to dermatochalasis

Measurements

1. **Margin–reflex distance** is the distance between the upper lid margin and the corneal reflection of a pen torch held by the examiner, at which the patient is directly looking (Fig. 1.115). The normal is 4–4.5 mm.
2. **Vertical fissure height** is the distance between the upper and lower lid margins, measured in the pupillary plane (Fig. 1.116). The upper lid margin normally rests about 2 mm below the upper limbus and the lower 1 mm above the lower limbus. This measurement is less in males (7–10 mm) than in females (8–12 mm). Unilateral ptosis can be quantified by comparison with the contralateral side. Ptosis may be graded as mild (up to 2 mm), moderate (3 mm) and severe (4 mm or more).
3. **Levator function** (upper lid excursion) is measured by placing a thumb firmly against the patient's brow to negate the action of the frontalis muscle, with the eyes in downgaze (Fig. 1.117a). The patient then looks up as far as possible and the amount of excursion is measured with a rule (Fig. 1.117b). Levator function is graded as normal (15 mm or more), good (12–14 mm), fair (5–11 mm) and poor (4 mm or less).

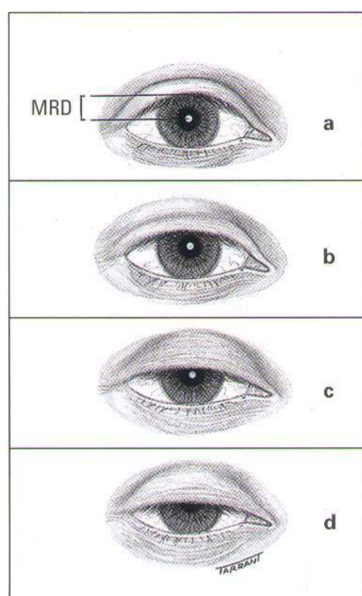


Fig. 1.115
Margin–reflex distance. (**a**) Normal; (**b**) mild ptosis; (**c**) moderate ptosis; (**d**) severe ptosis

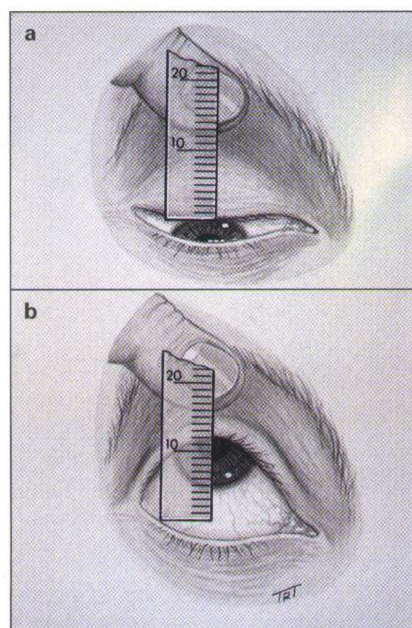


Fig. 1.117
Measurement of levator function

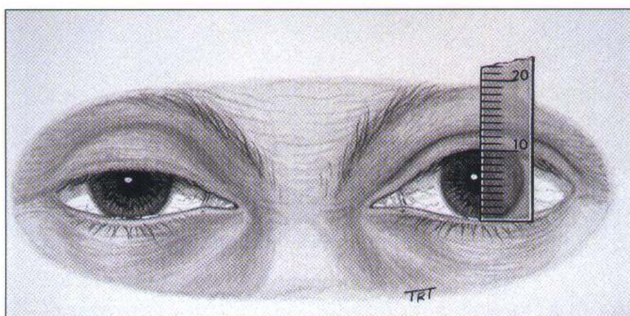


Fig. 1.116
Measurement of vertical fissure height



Fig. 1.118
Upper lid crease

4. **Upper lid crease** is the vertical distance between the lid margin and the lid crease in downgaze (Fig. 1.118). In females it measures about 10 mm and in males 8 mm. Absence of the crease in a patient with congenital ptosis is indirect evidence of poor levator function, whereas a high crease suggests an aponeurotic defect. The skin crease is also used as a guide to the initial incision.
5. **Pretarsal show** is the distance between the lid margin and the skin fold with the eyes in the primary position.

Associated signs

1. **Increased innervation** may flow to the levator muscle of a unilateral ptosis, particularly in upgaze. Associated increased innervation to the contralateral normal levator will result in lid retraction (Fig. 1.119). The examiner should therefore manually elevate the ptotic lid and look for a droop of the opposite lid. If this occurs, the patient



Fig. 1.119
Left ptosis resulting in right lid retraction

should be warned that surgical correction may induce a drop in the opposite lid.

2. **Fatiguability** is tested by asking the patient to look up without blinking for 30 seconds. Progressive drooping of one or both lids, or inability to maintain upgaze, is suggestive of myasthenia (Fig. 1.120). Myasthenic ptosis may show an overshoot of the upper lid on saccade from downgaze to the primary position (Cogan twitch sign) and also a 'hop' on sidegaze.
3. **Ocular motility defects**, particularly of the superior rectus, must be evaluated in patients with congenital ptosis. Correction of an ipsilateral hypotropia may improve the degree of ptosis.
4. **Jaw-winking** phenomenon can be detected by asking the patient to chew and move the jaws from side to side (*see below*).
5. **Bell phenomenon** is tested by manually holding the lids open, asking the patient to try to close the eyes and observing upward rotation of the globe. A weak Bell phenomenon carries a risk of postoperative exposure keratopathy, particularly following large levator resections or suspension procedures.



Fig. 1.120
Increase in ptosis on fatigue testing

Marcus Gunn jaw-winking syndrome

About 5% of all cases of congenital ptosis manifest the Marcus Gunn jaw-winking phenomenon. The vast majority of cases are unilateral. Although the exact aetiology is unclear, it has been postulated that a branch of the mandibular division of the fifth cranial nerve is misdirected to the levator muscle.

Signs

- Retraction of the ptotic lid in conjunction with stimulation of the ipsilateral pterygoid muscles by chewing, sucking, opening the mouth (Fig. 1.121) or contralateral jaw movement (Fig. 1.122).
- Less common stimuli to winking include jaw protrusion, smiling, swallowing and clenching of teeth.
- Jaw-winking does not improve with age, although patients may learn to mask it.

Treatment

This should be considered if jaw-winking or ptosis represents a significant functional or cosmetic problem. Although no surgical treatment is entirely satisfactory, possible approaches include:

1. **Unilateral levator resection** for mild cases with levator function 5 mm or better.
2. **Unilateral levator disinsertion** and part resection with ipsilateral brow (frontalis) suspension for more severe cases.
3. **Bilateral levator disinsertion** and part resection with bilateral brow suspension to produce a symmetrical result.

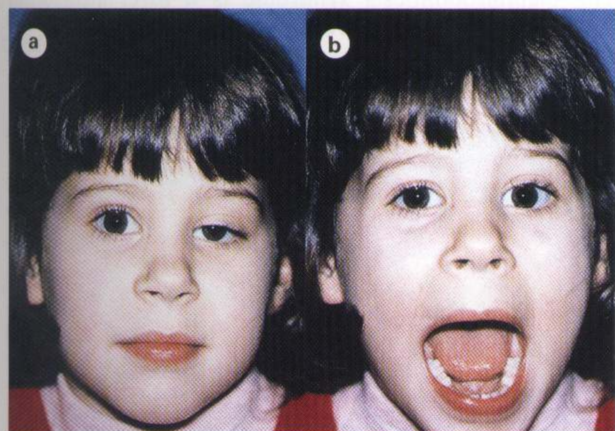


Fig. 1.121
Marcus Gunn jaw-winking syndrome. **(a)** Normal position showing a moderate left ptosis; **(b)** retraction of left eyelid on opening the mouth

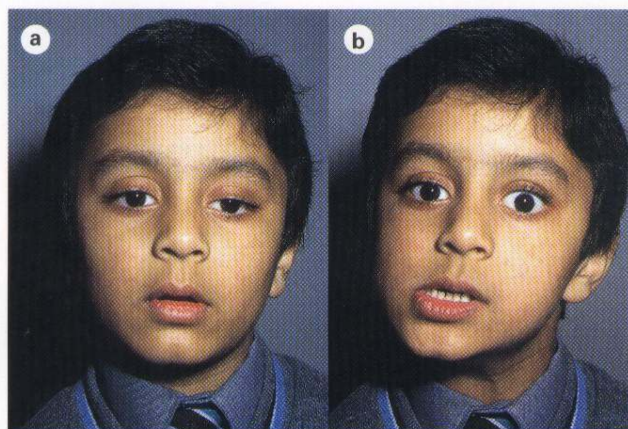


Fig. 1.122
Marcus Gunn jaw-winking syndrome. **(a)** Normal position showing a mild left ptosis; **(b)** retraction of the left eyelid on moving the jaw to the contralateral side

Third nerve misdirection

Third nerve misdirection syndromes may be congenital or, more frequently, follow acquired third nerve palsies.

1. **Signs**. Bizarre movements of the upper lid which accompany various eye movements (Fig. 1.123).
2. **Treatment** is by levator disinsertion and brow suspension.

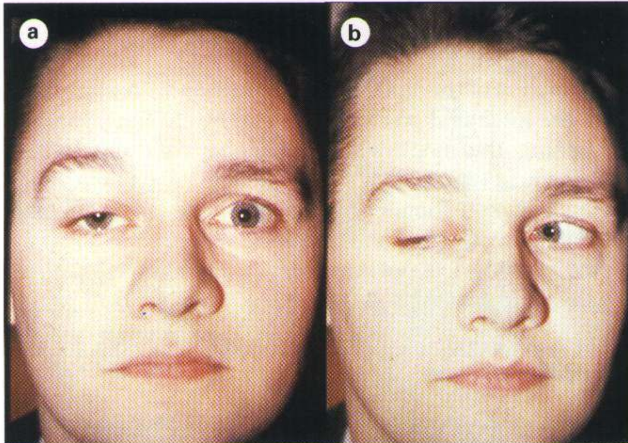


Fig. 1.123
Third nerve misdirection. (**a**) Normal position showing a moderate right ptosis; (**b**) worsening of ptosis on right gaze (Courtesy of S. Vardy)

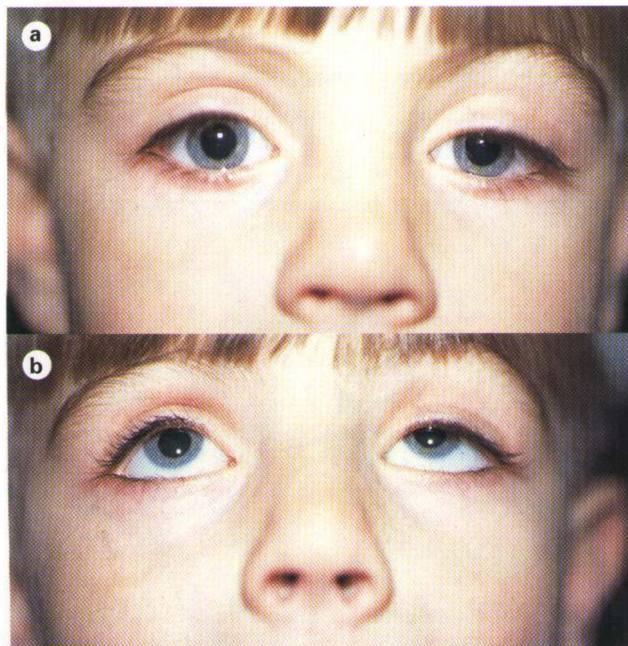


Fig. 1.124
(**a**) Moderate left congenital ptosis with absent lid crease; (**b**) poor levator function

Simple congenital ptosis

Simple congenital ptosis is probably caused by failure of neuronal migration or development with muscular sequelae. A minority of cases are hereditary.

Signs

- Unilateral or bilateral ptosis of variable severity.
- Absent upper lid crease and poor levator function (Fig. 1.124).
- In downgaze the ptotic lid is higher than the normal because of poor relaxation of the levator muscle. This is in contrast to acquired ptosis in which the affected lid is level with or lower than the normal on downgaze.

> *NB:* Following surgical correction the lid lag in downgaze may worsen (Fig. 1.125).

Associations

- Superior rectus weakness may be present because of its close embryological association with the levator.
- Compensatory chin elevation in severe bilateral cases (Fig. 1.126).
- Refractive errors are common and more frequently responsible for amblyopia than the ptosis itself.

Treatment

This should be carried out during the preschool years when accurate measurements can be obtained, although it may be considered earlier in severe cases to prevent amblyopia. Most cases require levator resection.

Blepharophimosis syndrome

The blepharophimosis syndrome is a rare, autosomal dominant, congenital disorder.



Fig. 1.125
Left lid is higher than the right following surgery for congenital ptosis



Fig. 1.126
Compensatory chin elevation for severe bilateral congenital ptosis



Fig. 1.127
Blepharophimosis syndrome

1. Signs (Fig. 1.127)

- Moderate to severe symmetrical ptosis with poor levator function.
- Short horizontal palpebral aperture.
- Telecanthus and epicanthus inversus.
- Lateral ectropion of lower lids.
- Poorly developed nasal bridge and hypoplasia of the superior orbital rims.

2. Treatment initially involves correction of epicanthus and telecanthus followed a few months later by bilateral frontalis suspension. It is also important to treat amblyopia, which is present in about 50% of cases.

Aponeurotic ptosis

Aponeurotic ptosis is caused by dehiscence, disinsertion or stretching of the levator aponeurosis which restricts transmission of force from a normal levator muscle to the upper lid. It is most frequently caused by involutional age-related degenerative changes.

1. Signs (Fig. 1.128)

- Variable, usually bilateral ptosis with good levator function.
- High upper lid crease (12 mm or more) because the posterior attachments of the aponeurosis to the tarsus have detached whereas the anterior attachments to the skin have remained intact and pulled the skin crease superiorly.
- In severe cases the upper lid crease may be absent, the eyelid above the tarsal plate very thin and the upper sulcus deep (Fig. 1.129).



Fig. 1.128
(a) Moderate right involutional ptosis with brow over-action; **(b)** high right upper lid crease as compared to left; **(c)** good levator function



Fig. 1.129
Severe bilateral involutional ptosis with thinning of the lids above the tarsal plates and deep sulci



Fig. 1.130
Mechanical ptosis due to a neurofibroma

NB: Involutional ptosis may be confused with myasthenic ptosis because it frequently gets worse towards the end of the day. This is because of fatigue of the Müller muscle, which has to work harder to keep the lid elevated.

2. **Treatment** options include levator resection, reinsertion or anterior levator aponeurosis repair.

Mechanical ptosis

Mechanical ptosis is the result of impaired mobility of the upper lid. It may be caused by dermatochalasis, large eyelid tumours such as neurofibromas (Fig. 1.130), scarring, severe lid oedema and anterior orbital lesions.

Principles of surgery

Fasanella–Servat procedure

1. **Indications**. Mild ptosis with levator function of at least 10 mm. This includes most cases of Horner syndrome and very mild congenital ptosis.

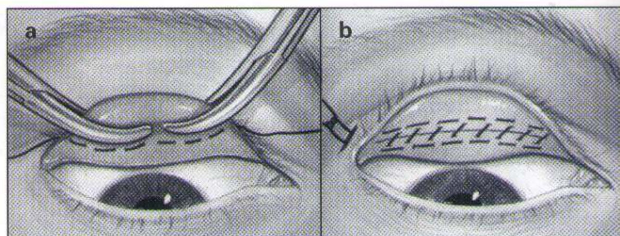


Fig. 1.131
Fasanella–Servat procedure

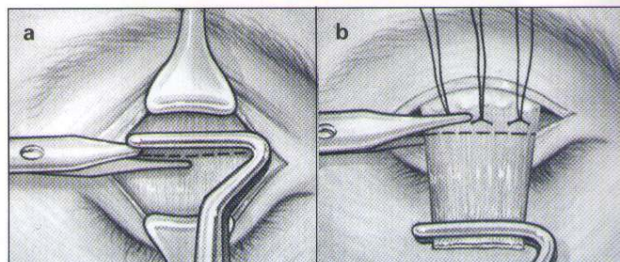


Fig. 1.132
Levator resection

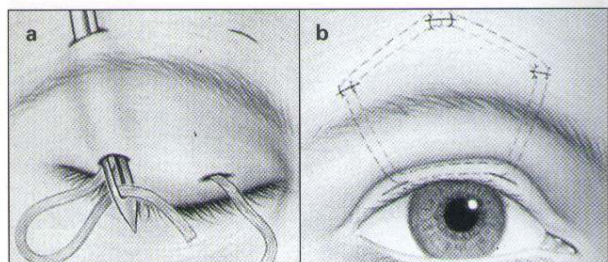


Fig. 1.133
Frontalis suspension

2. **Technique**. The upper border of the tarsus is excised together with the lower border of Müller muscle and overlying conjunctiva (Fig. 1.131).

Levator resection

1. **Indications**. Any ptosis, provided levator function is at least 5 mm. The amount of resection is determined by levator function and the severity of ptosis.
2. **Technique**. Shortening of the levator complex through an anterior (skin) or posterior (conjunctival) approach (Fig. 1.132).

Frontalis suspension

1. **Indications**
   - Severe ptosis (>4 mm) with very poor levator function (<4 mm).
   - Marcus Gunn jaw-winking syndrome.
   - Aberrant regeneration of the third nerve.

- Blepharophimosis syndrome.
- Total third nerve palsy.
- Unsatisfactory result from a previous levator resection.

2. **Technique**. Suspension of the tarsus from the frontalis muscle with a sling consisting of autologous fascia lata or non-absorbable synthetic material such as proline or silicone (Fig. 1.133).

Aponeurosis repair

1. **Indications**. Aponeurotic ptosis with excellent levator function.
2. **Technique**. Advancement and suturing of healthy aponeurosis to the tarsal plate through an anterior or posterior approach.

Miscellaneous acquired disorders

Dermatochalasis

Dermatochalasis is a very common, usually bilateral condition which typically affects elderly patients, characterized by redundant upper lid skin which may be associated with herniation of fat through a weak orbital septum (Fig. 1.134). The lids have a baggy appearance with indistinct lid creases (*see* Fig. 1.114). Treatment of severe cases, involves excision of redundant skin (blepharoplasty).

Blepharochalasis

Blepharochalasis is a rare condition characterized by recurrent episodes of painless, non-pitting oedema of both upper lids which usually resolves spontaneously after a few days. It usually starts around puberty and, with time, the episodes become less frequent. Severe cases may cause stretching of upper lid skin so that it becomes redundant and may acquire the appearance of wrinkled cigarette paper (Fig. 1.135). In other cases weakening of the orbital septum results in fat herniation.



Fig. 1.134
Fat herniation in dermatochalasis



Fig. 1.135
Left aponeurotic ptosis and very thin upper lid skin resulting from blepharochalasis

Floppy eyelid syndrome

Floppy eyelid syndrome is an uncommon, unilateral or bilateral condition, which is frequently misdiagnosed. It

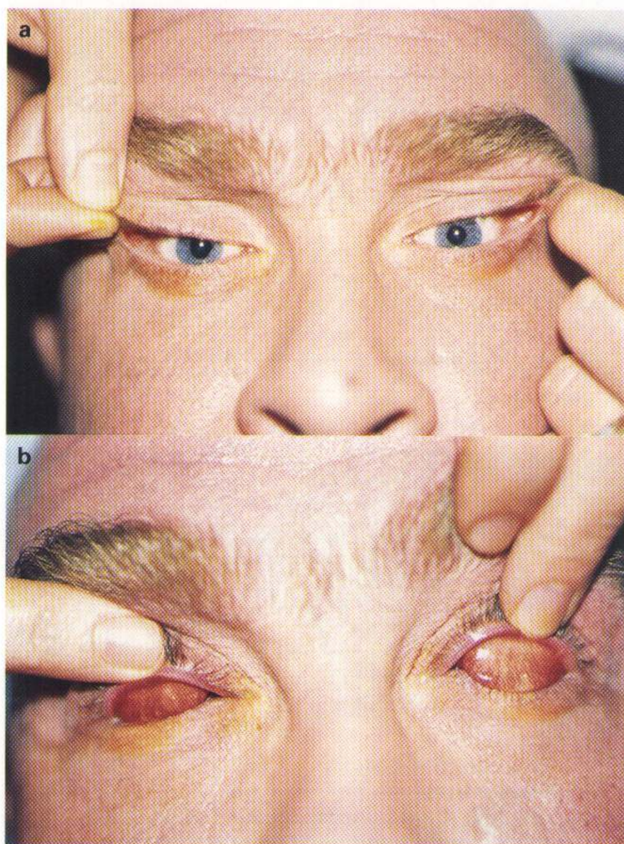


Fig. 1.136
Floppy eyelid syndrome. (**a**) Loose upper eyelids; (**b**) severe conjunctivitis of exposed areas

typically affects very obese men who may also suffer from sleep apnoea and snoring.

1. **Signs**
   - Rubbery and loose upper eyelids (Fig. 1.136a).
   - Eversion of the lids during sleep results in trauma to the exposed tarsal conjunctiva and chronic papillary conjunctivitis (Fig. 1.136b).
2. **Treatment** of mild cases involves nocturnal eye shields or taping of the lids. Severe cases require horizontal lid shortening.

Lid retraction

Lid retraction is suspected when the upper lid margin is either level with or above the superior limbus (Fig. 1.137). The classification is shown in Table 1.5.



Fig. 1.137
Bilateral lid retraction in thyrotoxicosis

Table 1.5 Causes of lid retraction

1. **Thyroid eye disease**
2. **Neurogenic**
   - contralateral unilateral ptosis
   - facial palsy due to unopposed levator action
   - third nerve misdirection
   - Marcus Gunn jaw-winking syndrome
   - Collier sign of the midbrain (Parinaud syndrome)
   - hydrocephalus
   - sympathomimetic drops
3. **Mechanical**
   - surgical overcorrection of ptosis
   - scarring of upper lid skin
4. **Congenital**
   - isolated
   - Duane retraction syndrome
   - Down syndrome
   - transient 'eye popping' reflex in normal infants
5. **Miscellaneous**
   - prominent globe (pseudo-lid retraction)
   - uraemia (Summerskill sign)

Miscellaneous congenital disorders

Epicanthic folds

Epicanthic folds are bilateral vertical folds of skin that extend from the upper or lower lid towards the medial canthus. They may give rise to a pseudo-esotropia.

1. **Signs**
   a. ***Palpebralis.*** The folds are symmetrically distributed between the upper and lower lid (Fig. 1.138). This is the most common type in Caucasians.
   b. ***Tarsalis.*** The folds originate in the medial aspect of the upper lid and extend medially before dissipating (Fig. 1.139). This is the most common type in Orientals.
   c. ***Inversus.*** The folds start in the lower lid and extend upwards to the medial canthal area. It is associated with the blepharophimosis syndrome (*see* Fig. 1.127).



Fig. 1.138
Epicanthus palpebralis and pseudo-esotropia



Fig. 1.139
Epicanthus tarsalis

d. *Superciliaris*. The folds arise above the brow and extend downwards to the lateral aspect of the nose.
2. **Treatment** of small folds is by Y-V plasty, whilst large folds require a Mustarde Z-plasty.

Telecanthus

Telecanthus is an uncommon condition characterized by increased distance between the medial canthi as a result of abnormally long medial canthal tendons (Fig. 1.140). It should not be confused with hypertelorism, in which there is wide separation of the orbits. Telecanthus may occur in isolation or it may be associated with the blepharophimosis syndrome.

1. **Systemic associations** include Waardenburg, Möbius, Treacher Collins, Rubinstein–Taybi and Turner syndromes.
2. **Treatment** is by shortening and refixation of the medial canthal tendons to the anterior lacrimal crests or a transnasal wire.

Epiblepharon

Epiblepharon is very common in Orientals and should not be confused with the much less common congenital entropion.

1. **Signs**. An extra horizontal fold of skin stretches across the anterior lid margin and the lashes are directed vertically, especially in the medial part of the lid (Fig. 1.141a). When the fold of skin is pulled down the lashes turn out and the normal location of the lid becomes apparent (Fig. 1.141b). In congenital entropion, however, the entire eyelid becomes pulled away from the globe.
2. **Treatment** is not required in the majority of cases because spontaneous resolution with age is the rule. Persistent cases are treated by excising a strip of skin and muscle, and fixation of the skin crease to the tarsal plate (Hotz procedure).



Fig. 1.140
Telecanthus

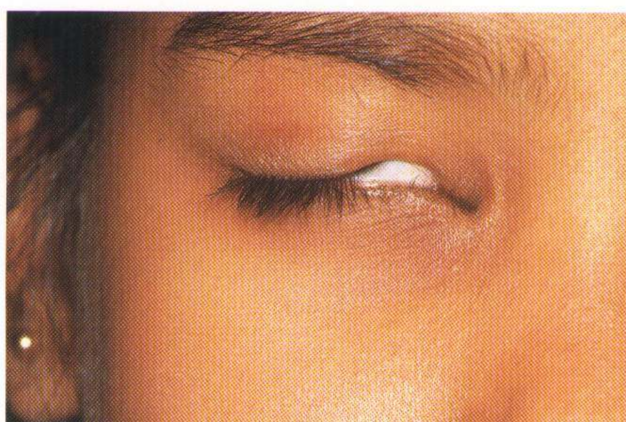


Fig. 1.142
Coloboma of the upper lid

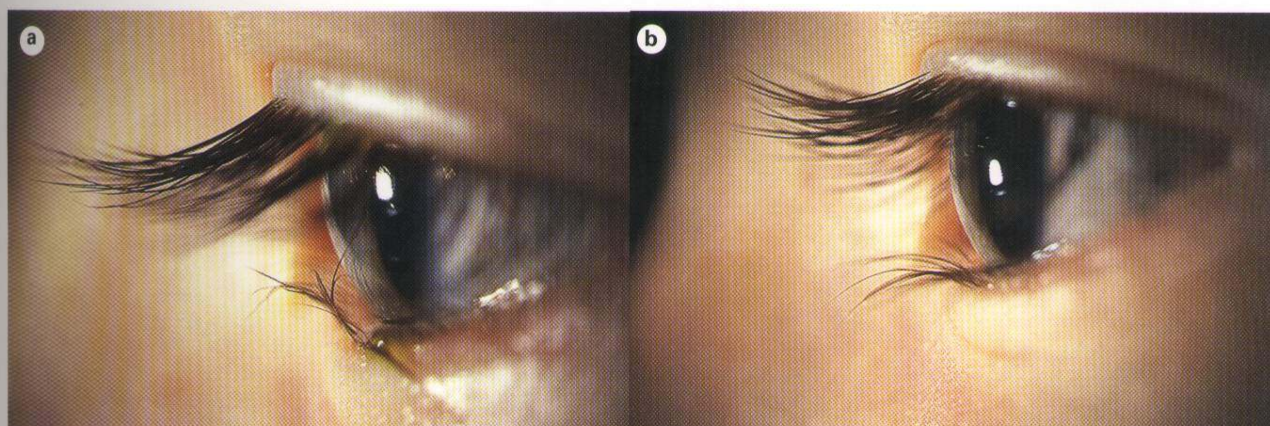


Fig. 1.141
(**a**) Epiblepharon; (**b**) normal position following manual correction


Fig. 1.143
Lower lid colobomas in Treacher Collins syndrome

Coloboma

A coloboma is an uncommon, congenital, unilateral or bilateral, partial or full-thickness eyelid defect.

1. **Upper lid** colobomas occur at the junction of the inner and middle thirds and are not associated with systemic anomalies (Fig. 1.142).
2. **Lower lid** colobomas occur at the junction of the middle and outer thirds and are frequently associated with systemic conditions such as Treacher Collins syndrome (Fig. 1.143).
3. **Treatment** of small to moderate defects is by primary closure, while large defects require skin grafts and rotation flaps.